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THE TROCHLEAR NERVE IN THE HUMAN EMBRYO AND FŒTUS

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IN a recent paper (Cooper, 1946 (*a*)) the development of the oculomotor and trochlear nuclei was described, and certain details of the origin and course of the oculomotor nerve were given. Since the trochlear nerve pursues an anomalous route to a decussation on the dorsum of the brain stem, further study was necessary before an adequate account could be submitted.

There are three questions relevant to the problem of the trochlear nerve :

1. Why is there a decussation ?

2. Why is the decussation on the dorsal aspect of the brain stem ?

3. How does the nerve reach the dorsal position where it decussates ?

Frazer (1928-1929) considered the problem from an embryological standpoint, and believed that the course of the nerve was affected by developmental occurrences in the region of the isthmus rhombencephali. He described and figured a displacement forwards

or invagination of the basal lamina of the hind-brain towards the mid-brain where it is folded down against the floor (basal) lamina of the latter producing an intra (basal)-laminar sulcus. The region where this occurs is the isthmus rhombencephali. Furthermore Frazer believed that the alar lamina extended on the outer side of the basal lamina of the isthmus, thus enabling the basal lamina which is displaced forwards, to form the lateral wall of the isthmus. In the 12 mm. embryo he found that where the basal lamina of the hind-brain continued rostrally to form the lateral wall of the isthmus, it produced a distinct rounded border in which was "situated the trochlear nucleus from which the nerve fibres run up (*i.e.*, dorsally) through the basal lamina to decussate." In the light of this rostral projection of the basal lamina, Frazer explained the deep course of the trochlear nerve, and stated that the nerve originally emerged from the basal lamina of the ventral aspect of the hind-brain. As the basal lamina is carried forwards to form the lateral wall of the isthmus, it takes the nerve with it. In this way the nerve gains the dorsal aspect of the isthmus where it emerges. He says "the issuing nerve is essentially ventral, although actually dorsal when the isthmus is thus completed."

Frazer attempted to explain the decussation of the trochlear nerves by likening them to the efferent spinal nerves which are composed mostly of homolateral fibres, but contain a few contralateral fibres, the latter of which decussate. He suggested that the fourth nerve consists entirely of contralateral fibres which decussate in the orthodox fashion.

Kappers, Huber and Crosby (1936) believe that the course of the trochlear nerve is due in part to a shifting forwards of the cells of origin during development. Hamilton, Boyd and Mossman (1945) state that the "atypical course of the fourth nerve, which apparently is to be regarded as somatic efferent in nature, has never been adequately explained."

From a study of a series of human embryos it became apparent that Frazer's explanation of the dorsal course of the trochlear nerves might profitably be reinvestigated. Regarding his suggested reasons for the decussation, it is relevant to refer to Gaskell's hypothesis. Reviewing the work of Beck, of Fürbringer and of Hoffman, Gaskell (1908) regarded the vertebrate eye muscles as comparable with the dorso-ventral prosomatic segmented muscles of the invertebrates. In the scorpion each of the pair of muscles known as the anterior dorso-plastron muscles crosses the mid-dorsal line to reach its insertion and carries its nerve supply with it. Gaskell suggested that these muscles become the superior oblique muscles of the vertebrates, and that herein lies the explanation of the trochlear route.

Present investigations

The trochlear complex as seen in a series of human embryos and fœtuses (4 mm. onwards) is described in the following pages. An account of the development of the isthmus rhombencephali is also included. The question how does the trochlear nerve reach the dorsal region of the brain stem is answered and the nature of the decussation is described. The investigation has not revealed why the decussation occurs or why it takes place on the dorsal aspect of the brain stem.

Since the publication of the paper referred to on p. 257, the author has obtained further early embryos in excellent condition. All embryos used in these investigations have been graded according to their length, and, where possible in accordance with the history. In some cases, however, the age estimate may not correspond strictly to the stage of development. For example in a 9 mm. and a 10 mm. embryo, recently received, both the oculomotor and trochlear nerves were found to have emerged from the brain stem. In a previous 10 mm. embryo neither was observed to have reached its superficial origin.

The earliest embryo available measures 4 mm. (estimated age between three and four weeks). Fore-, mid- and hind-brain are differentiated, and the optic vesicles form lateral projections from the fore-brain vesicle. A slight constriction exists between the rhombencephalon and mesencephalon which denotes the position of the future isthmus (Fig. 1). The otic vesicles appear in section as round cavities lying lateral to the hind-brain and separated from the surface ectoderm. On the cephalic aspect of each otocyst is the primitive acoustic ganglion composed of round or spindle-shaped cells with short processes which do not appear to extend either peripherally or centrally.

At a more rostral level is a larger ganglionic mass placed just caudal and lateral to the primary head vein (anterior cardinal vein). This is the trigeminal ganglion and, like the acoustic ganglion, it shows no connection with the neural tube. These ganglia indicate respectively the myelencephalic and metencephalic portions of the rhombencephalon. The thin roof plate of the rhombencephalon is present. The ganglia of the ninth and tenth cranial nerves and the posterior root ganglia of the spinal nerves are well formed, but none appears to be joined to the neural tube. It must be mentioned, however, that in such delicate tissue the continuity between the ganglia and the neural tube may have been destroyed as a result of death and histological preparation.

The neural tube consists throughout of ependymal, mantle and marginal layers. The mantle layer is similar in all parts, and

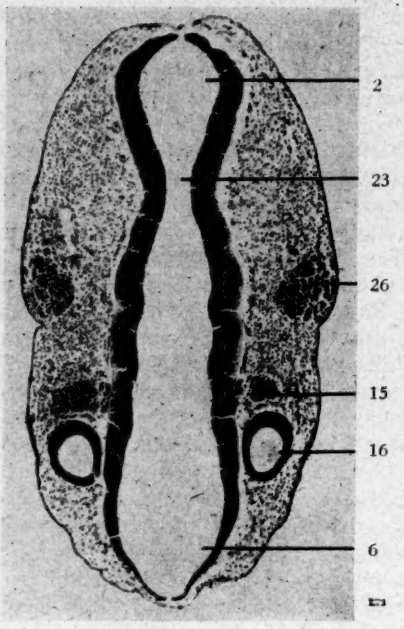

FIG. 1.

Section through head end of a 4 mm. embryo. ×50.

possesses seven to ten layers of undifferentiated (indifferent) cells. The roof (alar) and floor (basal) plates exhibit only the ependymal layer with one or two rows of cells. In no part of the neural tube is there any concentration of cells in the basal lamina indicative of efferent nuclear formation, and no efferent nerves, either spinal or cranial are recognisable.

An embryo of 9 mm. length (about $4\frac{1}{2}$ weeks) sectioned in the plane of the long axis of the rhombencephalon, gives an interesting picture of the developing brain stem. It will be seen from Fig. 2*a* that, owing to the neural flexures, the section illustrated in Fig. 2*b* shows the alar region of the metencephalon bounding the fourth ventricle in its cephalic half. Already a thickening of this alar plate has occurred on each side, forming the cerebellar rudiments. The thickening affects the two lateral portions of the alar plate, and each forms an intraventricular projection. As the fourth ventricle narrows at its cephalic extremity, the two (thickened)

cerebellar plates become approximated but do not meet, a small unthickened median portion of the (cerebellar) alar plates persisting at the apex of the fourth ventricle. This thin median plate becomes the superior medullary velum and Fig. 2*c* shows its continuity at the rostral apex of the fourth ventricle with the alar plate of the mesencephalon, a tangential section only of the latter being

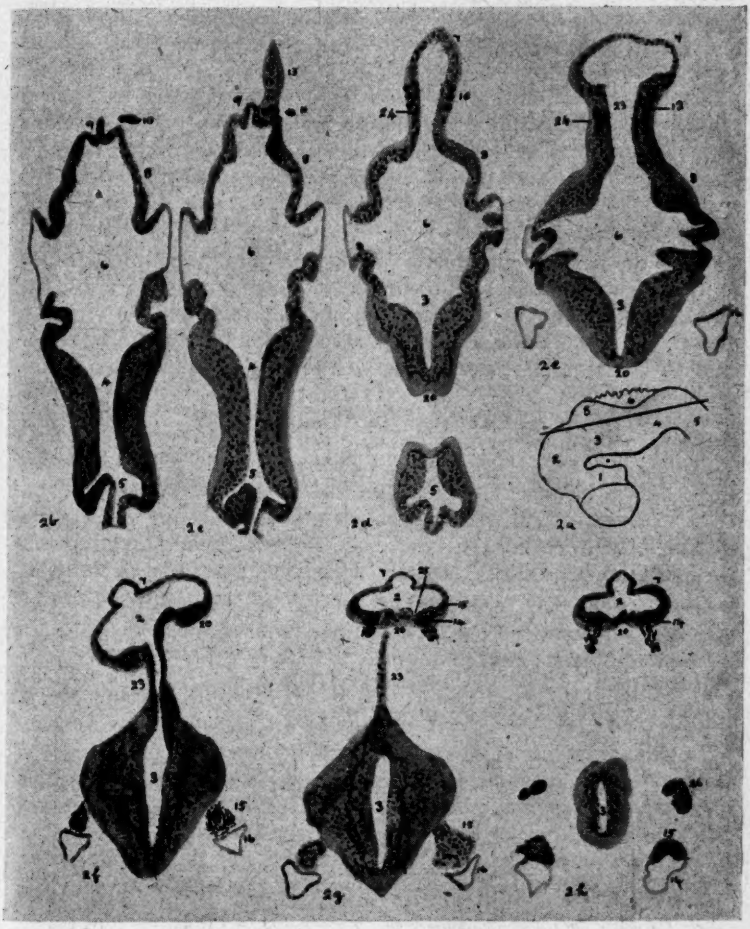

FIG. 2.

Serial sections through the brain stem of a 9 mm. embryo.

included. It is at the junction of the median portions of the metencephalic and mesencephalic alar plates that the decussation of the trochlear nerves is seen (Fig. 2c).

In the sections still deeper into the rhombencephalon, the opening up of the apical recess of the fourth ventricle into the wider cavity of the mesencephalon is visible (Fig. 2d). The narrow communication between the metencephalon and mesencephalon is the isthmus. Another point of interest is apparent in Fig. 2d. Here the lateral alar thickenings of the metencephalon (which form the cerebellar plates) continue rostrally side by side, and in this way become continuous with the lateral walls of the narrow isthmus. In each lateral wall the trochlear nerve can be identified lying on the outskirts of the mantle layer. Subsequent sections at more ventral levels show that the lateral walls of the isthmus finally merge into the basal plate of the mesencephalon where the narrow isthmic channel gives place to the wider mesencephalic cavity (Figs. 2e, f, g and h).

Thus the rostral extremities of the lateral walls of the isthmus join the floor or basal plate of the mesencephalon, forming the lateral parts. The intra (basal)-laminar sulci, described by Frazer, demarcate the superadded lateral parts of the floor plate from the median part which corresponds to the true basal lamina of the mid-brain.

Thus in this 9 mm. embryo there can be seen :—

1. Metencephalic alar thickenings on each side of the cephalic part of the fourth ventricle forming the cerebellar plates.

2. Mid-line apical alar region not thickened, but joining the rostral extremities of the cerebellar plates together and forming the superior medullary velum. Here the trochlear decussation occurs.

3. Traced rostrally, the metencephalic alar thickenings continue as the lateral walls of the isthmus.

4. Still more rostrally, the lateral walls of the isthmus merge into the floor plate of the mesencephalon.

In this series it is apparent that in the lateral walls of the isthmus there is continuity between tissue derived from metencephalic alar and mesencephalic basal plates. This junctional tissue forms the lateral isthmic walls, while the continuity of the superior medullary velum and the mesencephalic alar lamina closes the isthmus dorsally. Owing to the plane of the sectioning, the floor of the isthmus is not seen.

The nucleus of the trochlear nerve is recognisable as a small group of slightly larger differentiating cells lying on the outskirts

of the mantle layer in the lateral wall of the isthmus (*Fig. 2e*). The lateral isthmic walls blend with the mesencephalic basal plate and form the lateral parts of its floor, and here the oculomotor nuclei are situated. Thus the oculomotor and trochlear nuclei are in series, but at this 9 mm. stage, the former is located in the mesencephalon and the latter in the isthmus. From the trochlear nucleus a tiny nerve bundle runs along the edge of the mantle layer and can be followed in the lateral wall of the isthmus in a caudal direction to the apex of the fourth ventricle where the decussation occurs in the superior medullary velum and the nerve gains its superficial origin (Figs. 2*e*, *d* and *c*).

Sectioning of an embryo of 13 mm. length in the coronal plane from the vertebral column forwards, yielded a dorso-ventral or alar-basal series of the brain stem (Fig. 3*a*). It will be seen that the basal plate of the metencephalon forms the floor of the fourth ventricle, and the alar region, thickened by the intra-ventricular cerebellar plates, bounds the ventricle laterally (*Fig. 3b*). The cerebellar rudiments exhibit differentiation, and ependymal, mantle and marginal layers are wide and distinct. The thick cerebellar plates incline towards each other and towards the mid-line, but do not meet. At the cephalic apex of the fourth ventricle they are linked by alar tissue not included in the cerebellar thickening. This thin tissue is the superior medullary velum (*Fig. 3b*). Sections at a more cephalic level contain the decussation of the trochlear nerves in the velum and their emergence on the dorsal surface of this region (Fig. 3*c*). The more rostral part of the decussation occurs where the superior medullary velum is meeting the alar plate (tectum) of the mesencephalon (Figs. 3c and 4).

Traced in the cephalic direction, the two lateral alar (cerebellar) plates of the metencephalon, which are separated from each other at the apex of the fourth ventricle by the superior medullary velum, remain distinct, but lie side by side, leaving a slit-like cavity between them as the mid-brain is approached. The region with the narrow cavity is the isthmus, and the rostral continuation of each metencephalic alar (cerebellar) plate comes to form a lateral wall of the isthmus (*Fig. 3d*).

The narrowing of the isthmic cavity is the result of the close approximation of the lateral walls. In consequence, the basal lamina of the isthmus is compressed so that part of it actually forms the most ventral part of the lateral isthmic wall. But the basal lamina of the isthmus is continuous rostrally and caudally with that of the mesencephalon and metencephalon respectively, although in both these regions it is expanded by the widening of the cavities. The elongation of the isthmic cavity is exaggerated owing to obliquity of the sections.

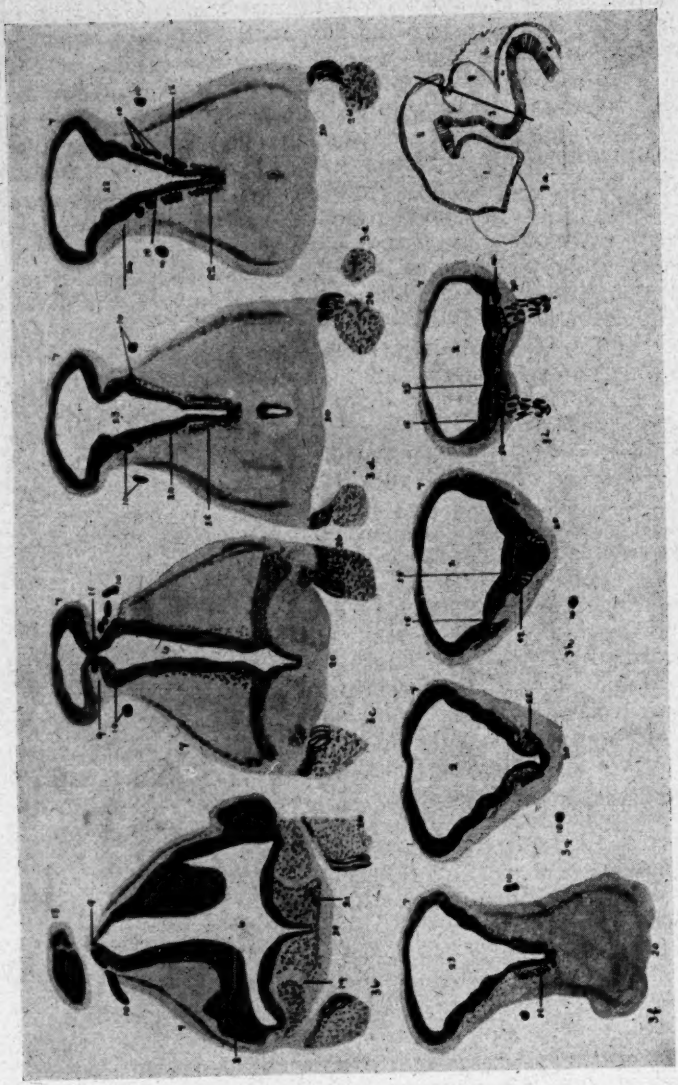

FIG. 3.

Serial sections through the brain stem of a 13 mm. embryo.

At a more cephalic level the cavity of the isthmus joins the wider cavity of the mesencephalon, and the lateral walls of the isthmus merge into the basal plate of the mid-brain forming its outer parts. The most ventral part of the lateral wall of the isthmus is really basal lamina (Fig. 3*e*), and here is situated the trochlear nucleus in series with the oculomotor nucleus (Figs. 3*e* and 5). Thus the trochlear nucleus lies in the lateral isthmic wall, but this

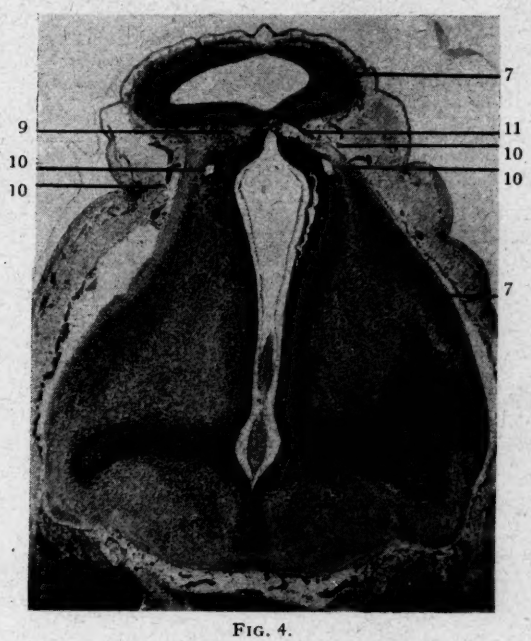

FIG. 4.

Region of isthmus of a 13 mm. embryo. (See Fig. 3*c*). ×30.

is only because the basal lamina has been pushed into this position by the elongation of the lateral isthmic walls and consequent narrowing of the contained cavity.

From the trochlear nucleus a bundle of fibres runs dorsally in the lateral walls of the isthmus and is then carried caudally to the apex of the fourth ventricle where it decussates with its fellow in the superior medullary velum and emerges superficially (Figs. 3*e*, *d*, *c* and *b*).

From this series of a 13 mm. embryo it would appear that there is a continuity of tissue between the cerebellar (alar) plates of the

metencephalon, the lateral wall of the isthmus, and the floor (basal) plate of the mesencephalon. This continuity ultimately enables the cerebello-mesencephalic fibres (superior cerebellar peduncles) to follow the route they do, and in the embryo of 19 mm. these fibres have traversed this pathway into the basal plate of the mid-brain where they decussate (Cooper, 1946 (*b*)). This then

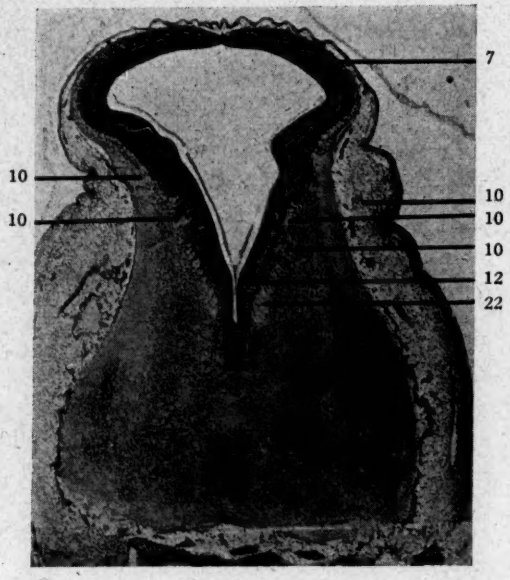

FIG. 5.

Cephalic end of isthmus of a 13 mm. embryo. (See Fig. 3*e*). ×30.

is the essential difference between Frazer's account of the development of the isthmus and the present findings. He described the lateral wall of the isthmus as being formed by a thrust forwards of the basal lamina of the metencephalon towards the mesencephalon. The present investigation seems to show that the lateral wall of the isthmus unites the rostral continuation of the metencephalic alar (cerebellar) plate to the mesencephalic basal plate, and thus provides a route for the cerebello-mesencephalic fibres.

An embryo of 15 mm. sectioned from the highest part of the mesencephalon gives another view of the isthmus and the trochlear nerves (Fig. 6*a*). In the sections through the caudal extremity of the metencephalon, the alar and basal plates are lying in the

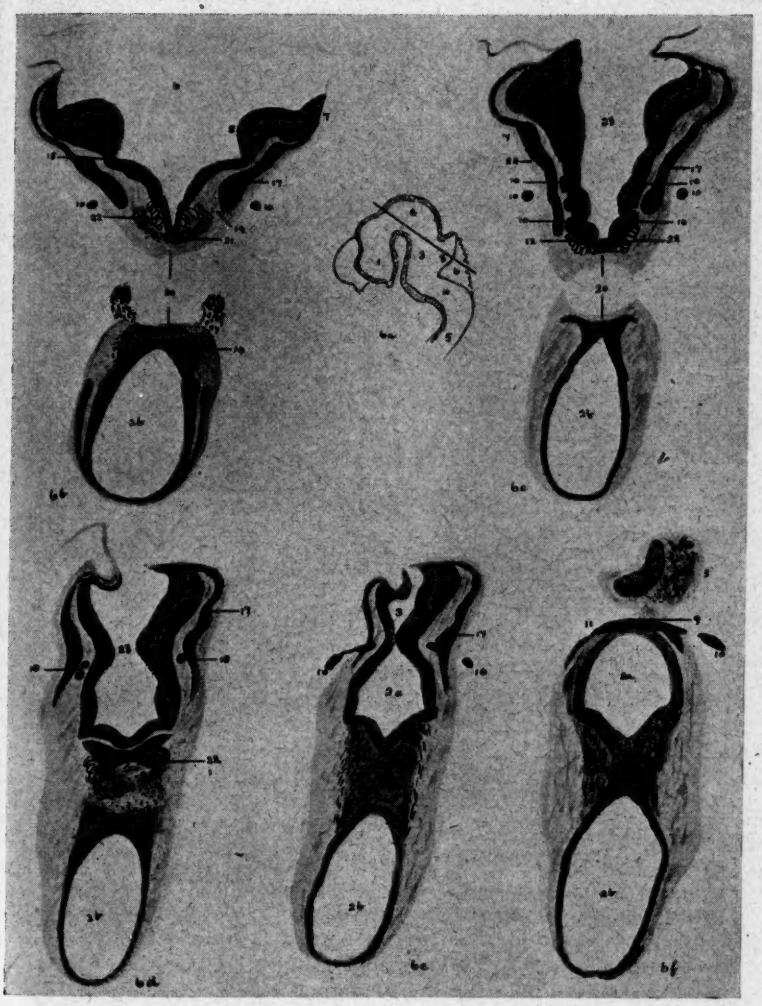

FIG. 6.

Serial sections through the brain stem of a 15 mm. embryo.

floor of the fourth ventricle, the roof being ependymal (Fig. 6*b*). The proliferation of neuroblasts is greater in the alar than in the basal plates, and in the former a dorsally directed intra-ventricular enlargement forms the cerebellar rudiment. In addition a spread of cells from the alar portion has progressed ventro-medially towards the basal plate. The basal portion has given rise to a cell

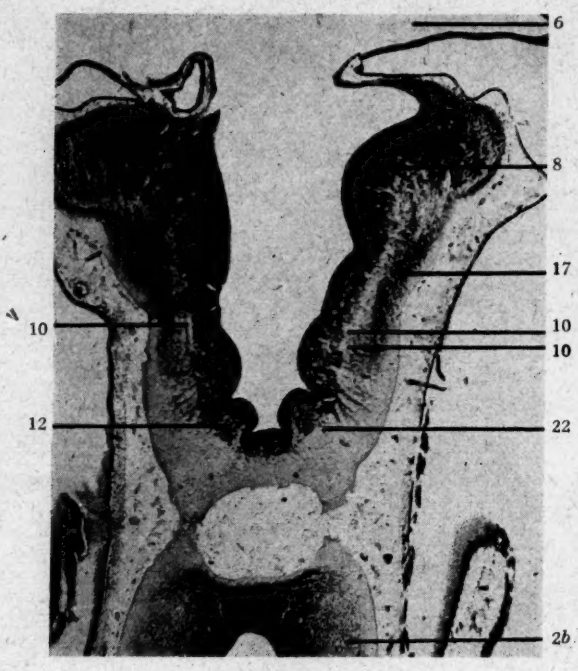

FIG. 7.

Region of isthmus of a 15 mm. embryo. (See Fig. 6c). ×30.

spread which is limited to the median part of the floor of the fourth ventricle. These alar and basal spreads appear to be separated by a relatively cell-free zone sufficiently well defined to justify the assumption that it is an inter-laminar zone (Fig. 6*b*). It does not correspond, however, to the indentation on the ependymal aspect which is usually called the interlaminar sulcus (sulcus terminalis). It is suggested, therefore, that the junction of the alar and basal plates corresponds to the cell-free zone and not to the interlaminar sulcus. If this be true, then the alar plate is thickened

in its larger lateral part to form the cerebellar rudiment, while its smaller medial part adjacent to the basal plate is not enlarged. In other words the interlaminar sulcus is a depression resulting from the cerebellar enlargement of the lateral part of the alar plate.

Following the sections serially in a dorsal direction, it is found that the alar plates gradually approach each other until they lie parallel and bound a narrow cavity (Figs. 6*c* and 7). This region corresponds to the isthmus rhombencephali. The alar plates come to form the lateral walls of the isthmus, while the basal plate is pressed down to bound the ventral recess of the isthmic cavity. Furthermore, since the recess is exceedingly narrow, part of the basal plate assumes a lateral position and comes to lie in the most ventral part of the lateral isthmic wall, the rest of this wall being formed by alar plate (Figs. 6*c* and 7).

It is in this position that the trochlear nucleus is found, lying in the basal lamina of the isthmus, but where this lamina is forming the most ventral part of the lateral isthmic wall (Figs. 6*c* and 7). Traced more dorsally (Figs. 6*d* and *e*, and 8) the two lateral isthmic walls finally meet, thus separating off an overhanging portion of the metencephalic cavity from the isthmic cavity which now begins to widen out to join the mesencephalic cavity. During this widening out process, the isthmic basal lamina is spread out again, and once more lies in the floor plate when the mesencephalon proper is reached (Fig. 6*f*).

It is apparent from Figs. 6*e* and *f*, and 8 that the meeting of the lateral isthmic walls coincides with the superior medullary velum which continues into the alar (roof) plate of the mesencephalon. Also it will be seen that the lateral walls of the isthmus are continuous with alar tissue which forms the cerebellar plates more caudally. Furthermore owing to the mesencephalic flexure, the mesencephalic basal lamina comes into line with the lateral wall of the isthmus and this continues into the lateral part of the basal lamina of the mid-brain (Fig. 6*a*).

The trochlear nucleus in this series appears as a semilunar mass of neuroblasts lying in the compressed basal plate at the ventral extremity of the lateral isthmic wall. The neuroblasts are distinctly larger than any other cells (Figs. 6*c* and 7). From the cells in the nucleus, fibres turn dorsally and immediately enter the lateral isthmic wall proper which is mainly alar in origin (Figs. 6*c* and 7). Thence the nerve passes in the lateral wall of the isthmus to that position where the two lateral walls meet at the superior medullary velum. Here the two nerves decussate and emerge at their superficial origin (Figs. 6*e* and *f*, and 8).

Thus, judging from serial sections of early embryos cut :—

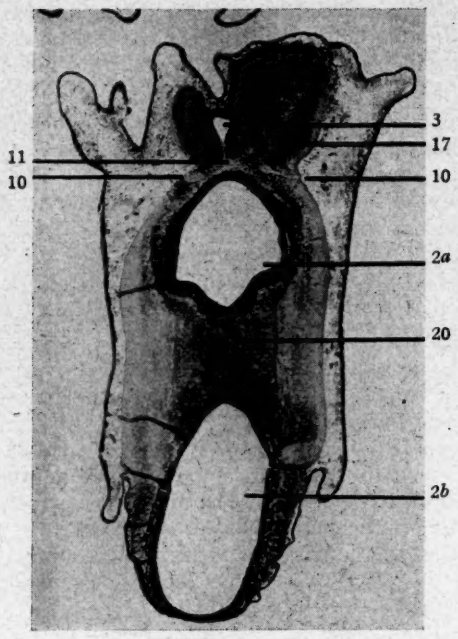

FIG. 8.

Cephalic end of isthmus of a 15 mm. embryo. (See Fig. 6e). ×20.

1. in the plane of the long axis of the rhombencephalon;

2. in the dorso-ventral (alar-basal) plane; and

3. from the highest part of the mesencephalon, the following points are demonstrable:—

1. The metencephalic alar tissue is continued rostrally in the lateral isthmic walls to the mesencephalic basal plate. (By the 19 mm. stage this route is traversed by cerebello-mesencephalic fibres (superior cerebellar peduncles)).

2. The trochlear nucleus lies in the lateral isthmic wall, but in the most ventral part which is formed by the basal plate. This plate is squeezed into this position by the alar tissue which forms the bulk of the lateral wall of the isthmus.

3. The trochlear fibres pass dorsally and caudally within the lateral wall of the isthmus to the alar plate of the metencephalon, these two structures being continuous. Thus the trochlear nerve

reaches the dorsum of the isthmus and decussates in the superior medullary velum and escapes on the dorsal aspect of the brain stem.

4. The position where the lateral isthmic walls join the mesencephalic basal lamina is marked by the intra (basal)-laminar sulcus.

5. In the metencephalon the line of demarcation between the alar and basal plates apparently does not correspond to the interlaminar sulcus, but it is indicated by a cell free zone.

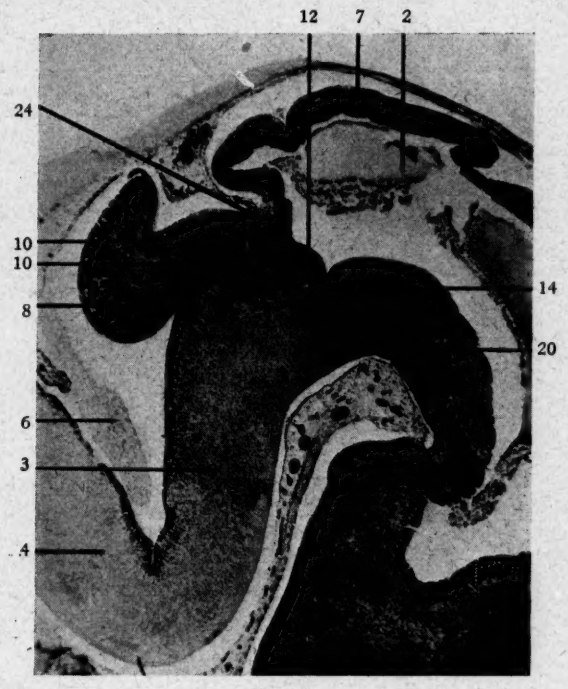

FIG. 9.

Parasagittal section through the brain stem of a 22 mm. embryo. ×25.

Figure 9 is a parasagittal section through the brain of an embryo of 22 mm. It has passed through the lateral wall of the isthmus. The fibres of the superior cerebellar peduncles sweep forwards and ventrally through the lateral wall of the isthmus to the basal plate of the mid-brain where they decussate and join the red nucleus. The oculomotor nucleus is recognisable in the tegmental (basal) region of the mid-brain. Just caudal to it is the trochlear nucleus lying in the basal plate and overhung by the enlarging

tectum of the mid-brain. Fibres of the trochlear nerve are seen to be running from the nucleus dorsally and caudally, and by following the direction of the cerebello-mesencephalic fibres, reach their decussation in the superior medullary velum.

In this series the nucleus of the trochlear nerve is apparently located in the isthmus in embryos up to 22 mm. length, but one point must be borne in mind. An accurate estimation of the extent of the isthmus is difficult owing to the modification produced by the neural flexures as age increases, and the resulting variation in the planes of serial sections.

The above observations provide a possible answer to the question how does the trochlear nerve reach its dorsal decussation? It has been shown that the trochlear nucleus at first lies in the isthmus, and the nerve issuing therefrom gains the dorsal region of the metencephalon in tissue which extends between the mesencephalic basal and metencephalic alar plates. It would seem that this is a route offering little resistence to the course of the nerve, and it also becomes the pathway for the fibres of the superior cerebellar peduncles. If the nerve pursued a ventral course it would ultimately need to penetrate through the fibres of the superior cerebellar peduncles which soon form a dense mass in the basal plate of the mesencephalon.

The next point to be considered is the final orientation of the trochlear nucleus in the caudal region of the mesencephalon. From Fig. 9 it will be seen that already the tectal (alar) plate of the mesencephalon has grown to a greater extent than the basal, and that it is overhanging the fore part of the isthmus. It is evident that when the extra-ventricular growth of the cerebellum occurs, it encroaches on the isthmus from behind. Examination of the adult brain stem justifies the statement that growth of the isthmus lags behind that of the mesencephalon and metencephalon, and its identity is somewhat obscured. It may be that the enlarging mesencephalon tends to draw the isthmus in the rostral direction, while the metencephalic development pushes it forwards. In addition the sweep of the fibres of the superior cerebellar peduncles from the metencephalon to the mesencephalon which commences at the 19 mm. stage, may carry the trochlear nucleus to a more cephalic position. As development proceeds, decrease in the degree of the mesencephalic and pontine flexures, which affect the basal plates, will help to establish the trochlear nucleus in a more cephalic position relative to the enlarging mesencephalic tectum.

The last point to be mentioned is the nature of the trochlear decussation. In all embryonic, fœtal and adult specimens examined, the trochlear nerve seems to form a compact bundle as the fibres emerge from the nucleus. This bundle pursues its dorso-caudal

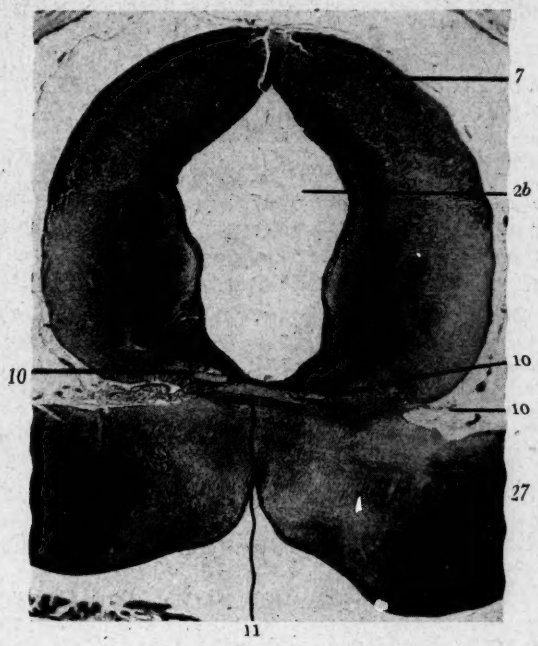

FIG. 10.

Section through the trochlear decussation of a 37 mm. embryo. ×20.

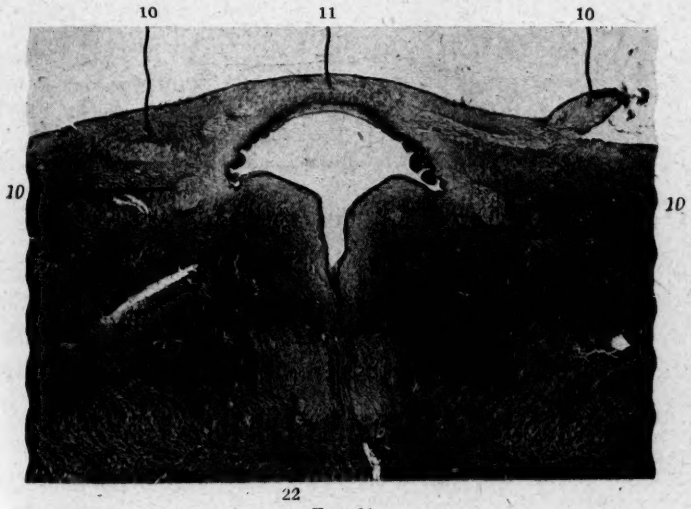

FIG. 11.

Section through the trochlear decussation of an adult. ×10.

course and a simple crossing of the two bundles occurs. There does not appear to be the thorough intermingling of fibres as in the great fibre decussations of the superior cerebellar peduncles, pyramids or medial lemnisci. Invariably a single bundle emerges at the surface (Figs. 4, 8, 10 and 11).

Conclusions

1. The two metencephalic alar plates (cerebellar rudiments) approximate closely to each other and to the mid-line at the rostral apex of the fourth ventricle. Here they are linked together by a thin stratum of tissue which becomes the superior medullary velum.

2. These metencephalic alar plates are continued rostrally around the lateral aspect of the neural tube at the position of the isthmus rhombencephali, and thus form its lateral walls.

3. Traced in the cephalic direction, the lateral isthmic walls join the basal plate of the mesencephalon on either side when the narrow cavity of the isthmus expands into the mid-brain cavity.

4. Thus a continuity of tissue exists between the metencephalic alar (cerebellar) plate, the lateral wall of the isthmus and the mesencephalic basal or floor plate. The fibres of the superior cerebellar peduncles follow this route from the cerebellum to the red nucleus at the 19 mm. stage of the embryo.

5. The trochlear nucleus originates in series with the oculomotor nucleus but in the basal plate of the isthmus. As the lateral walls of the isthmus link the metencephalic alar to the mesencephalic basal plates, the basal plate of the isthmus becomes compressed until it assumes a position at the ventral extremity of the lateral wall of the isthmus, and it is here that the trochlear nucleus is situated at first. The fibres from the nucleus form a bundle which turns dorsally at once and thus gains an alar situation in the lateral wall of the isthmus. The nerve then proceeds through the lateral wall of the isthmus caudally to the superior medullary velum where the decussation occurs.

6. Beyond 22 mm. (7 weeks) the trochlear nucleus is orientated in the mid-brain and suggestions are put forward to explain this apparent shifting forwards.

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KEY TO ILLUSTRATIONS.

1. Diencephalon.
2. Mesencephalon (*a*) caudal level; (*b*) cephalic level.
3. Metencephalon.
4. Myelencephalon.
5. Spinal cord.
6. Fourth ventricle and/or its taenial roof.
7. Alar plate.
8. Cerebellar rudiment.
9. Superior medullary velum.
10. Trochlear nerve.
11. Decussation of trochlear nerves.
12. Trochlear nucleus.
13. Tangential section of mesencephalic alar plate.
14. Oculomotor nucleus and/or nerve.
15. Acoustic ganglion.
16. Otocyst (otic vesicle).
17. Alar spread.
18. Interlaminar sulcus.
19. Interlaminar cell-free zone.
20. Basal plate.
21. Basal spread.
22. Medial longitudinal bundle.
23. Isthmus rhombencephali.
24. Lateral wall of isthmus.
25. Intra (basal)-laminar sulcus.
26. Trigeminal ganglion and nerve.
27. Superior cerebellar peduncle.

The use of a hand lens is recommended in examining the photographs.

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COLOUR VISION IN THE CONSULTING ROOM

BY

FRANK R. NEUBERT

GUERNSEY

IN 1941 an analysis of the colour-vision reports on 2,484 subjects suggested that the difference in the findings on colour-plates and colour-lanterns would form an interesting subject for investigation.

Such an investigation began, and during the following four years 43,395 subjects were examined: 40,380 males and 3,015 females.

Only two defectives (both unsafe) were found among the latter, and the final figures were restricted to the male sex only.

Many subjects were seen on whose colour sense opinions had differed, and an attempt was made to determine (*a*) why such defects could not be assessed easily and positively (*b*) why results on colour plates and colour lanterns did not always agree, and (*c*) whether an improvement could be made in clinical examination apart from the use of laboratory methods.

Colour Perception with Aviation Model

The terms " normal," " unsafe," " safe," " green-blind," and " red-blind," although scientifically inaccurate, are understood by all, and are used in their commonly accepted meaning in this résumé of the work done.

All the 40,380 men were asked to read the original Ishihara Colour Plates, 8th Edition, and each one who made a mistake suggesting a defective colour sense—2,235 in all—was investigated on a lantern (colour perception unit—aviation model) and by other experimental methods.

A group of 1,654 subjects was first examined on plates and lantern and the results compared.

TABLE I.

Examination of 1,654 previouslv untested subjects on Plates and Lantern.

		Ishihara Plates		Lantern Unit
Normal		1,585		1,623
Safe		20 (1·21%)		3 (0·015%)
Unsafe — Red-Blind	8	49 (3·1%)	3	28 (1·75%)
Unsafe — Green-Blind ...	38		22	
Unsafe — Red and Green-Blind	3		3	
Total		1,654		1,654

The difference between these findings was too great to be ignored, and a second group of 431 subjects, who were classified as unsafe by the plates, were examined on the lantern : 135 (31·3 per cent.) passed. From this it appeared that the plates were approximately one third more sensitive than the lantern in detecting defects in the colour sense.

TABLE II

Comparison of Defective Subjects by Ishihara Test and Lantern

Ishihara		Lantern	
Colour Defective Unsafe		Colour Defective Unsafe	Colour Defective Safe
Red-Blind ...	178	131	47
Green-Blind ...	236	153	83
Red & Green-Blind	17	12	5
Total	431	296 (68·7%)	135 (31·3%)

The manner in which the colour plates were read made it seem advisable to record the individual mistakes, and 1,573 unselected men were asked to read, without hesitation, a group of figures in an American series of plates (mentioned later).

686 (43·7 per cent.) of them were certainly normal or definitely defective. 532 (33·8 per cent.) of them made mistakes which were corrected at a second attempt. 355 (22.5 per cent.) of them made mistakes which were not corrected after two or more attempts, although such errors did not come into the category " Colour defective " according to the explanation book.

For example, if plates Nos. 1 and 2 (figures 89 and 43) were read as 89 and 45, altered to 89 and 48, then corrected to 89 and 43, the mistakes would appear due to observation rather than a defect in the colour sense and such cases were not classified as defective. This is in agreement with the instructions.

Pseudo-iso Plates

The original Ishihara plates began to fade, and tests were commenced with the series issued by the American Optical Company: The pseudo-isochromatic plates for testing colour perception.

Each series contains a pair of plates showing the numerals 45 and 73 which are intended to be similar: Ishihara Nos. 20 and 21, A.O.C. Nos. 33 and 34. These are supposed to be read only by the majority of the red-green blind, " but the majority of the normal and the totally colour-blind can hardly see them " (Ishihara 1939). The results of 11,348 readings were analysed.

By Ishihara standards 445 (3·9 per cent.) were definitely colour-blind, 370 (3·2 per cent. Group A) read the Japanese figures, but only 193 (1·7 per cent. Group B) read the American ones, a difference of 47 per cent.

TABLE III

Comparison of 445 defective subjects by Ishihara and American Plates.

	Ishihara Plates	American Plates
Read (unsafe)	370 (83·0%) Group A	193 (43·4%) Group B
Not read (safe)	75 (17·0%)	252 (56·6%)
Total	445	445

The 370 subjects (Group A) were examined on the lantern and 124 (33·5 per cent.) of them were found to be safe.

The 193 subjects (Group B) were examined similarly and 55 (29·5 per cent.) were found to be safe.

Examination of these results showed that, whereas only two subjects could read the American and not the Japanese figures, 200 were able to read the Japanese but not the American. The lantern results of these cases are compared in Table IV.

TABLE IV

Classification by Lantern of subjects in Table III who read Ishihara plates Nos. 20 and 21 and American Plates Nos. 33 and 34.

Lantern Classification	Ishihara Unsafe	American Unsafe
Safe ...	124 (33·5%)	55 (29·5%)
Unsafe ...	246 (66·5%)	138 (70·5%)
Total ...	370 (Group A)	193 (Group B)

This not inconsiderable difference emphasises the exceptional difficulty in producing dyes which are suitable for colour-vision testing.

When a British reprint of the Ishihara plates was issued, a comparison was made between it and the Japanese set, but as so many normal subjects read without much difficulty the figures which only the colour-blind were intended to see, the set was not included in the investigation and only the original series was used.

The Ishihara test claims to separate by means of plates Nos. 22, 23, 24, and 25, the " red-blind " from the " green-blind." A detailed examination on the lantern was made of those defective classified by this means.

TABLE V

Ishihara		Lantern Test			Passed Lantern Test
		Failed to recognise			
		Red only	Green only	Red and Green	
Red-Blind ...	116	7	53	35	21
Green-Blind ...	194	16	72	35	71
Red and Green-Blind	27	3	11	6	7
Total	337	26	136	76	99

Out of the Ishihara " red-blind " cases, only 7 failed to recognise only red on the lantern, whereas 35 were unable to recognise both red and green, and 21 passed the test.

Out of 194 who were Ishihara " green-blind," 72 were unable to recognise only green on the lantern, whereas 35 failed to recognise both red and green, and 71 passed the lantern test.

It has been claimed that any subject classified by these figures will eventually fail with the lantern if examined persistently, but this was not found to be the case.

TABLE VI

An analysis into "Safe" and "Unsafe" on the lantern of 368 subjects classified according to Ishihara plates as Colour Defective Unsafe.

	Red-Blind	Green-Blind	Red and Green-Blind	Total	
Ishihara Plates	122	223	23	368	
Lantern—Safe...	11	81	6	98	368
„ Unsafe	111	142	17	270	

A total of 368 cases was classified, of whom 122 were found to be red-blind and 223 were found to be green-blind. 92 of these cases passed the lantern test after having been examined persistently.

During these tests a comparison was made between the aviation model colour perception unit and the Edridge-Green colour lantern; 190 defective subjects were examined on each, using the technique outlined in the respective instructions.

The comparison suggests an inherent weakness in this type of lantern.

TABLE VII

The percentage comparison between the Aviation Model and Edridge-Green Lantern.

Unsafe Aviation Model	*Unsafe* Edridge-Green	47·4%
Unsafe Aviation Model	*Safe* Edridge-Green	15·8%
Safe Aviation Model	*Unsafe* Edridge-Green	15·8%
Safe Aviation Model	*Safe* Edridge-Green	21·0%

PART II

This examination and comparison of the usual clinical methods of determining the state of the colour sense suggested that more attention should be devoted to lantern tests and a considerable number of experimental lanterns were made. The original postulates of Guttmann, translated in Parsons' "Colour Vision," suggested the lines along which these experiments should develop.

Guttmann found that those with defective colour perception differed in seven respects from the normal.

(1) They required a smaller difference in the yellow but a greater difference in the green for discrimination.

(2) They were more dependent upon luminosity for hue discrimination.

(3) They developed a capacity for distinguishing differences in luminosity which they translated into hue.

(4) They required a larger area of retinal stimulation.

(5) They required a longer time to make their decision.

(6) Their colour sense was more readily fatigued (successive contrast).

(7) They had an increased sense of simultaneous contrast.

The chromatic threshold

Parsons states that the appreciation of colours only occurs with lights of moderate or high intensity (photopic vision). If a spectrum of low intensity is seen with the dark adapted eye it appears as a grey band, differing in brightness in different parts (scotopic vision).

It would appear from this that, with the very low threshold for red, an exaggeration between the appreciation of red and green, or between red and yellow, would be made by a defective person if two or three colours were presented simultaneously to the photopic eye by means of a lamp with controlled illumination. A rheostat was fitted to all lanterns.

Size of lantern aperture

According to Guttmann's fourth postulate, a colour defective person requires a considerably larger area of stimulation for the perception of hue.

The duplicity theory of vision asserts that the retinal cones are responsible for the perception of colour. The cones are most numerous at the fovea centralis, where rods are absent, and decrease proportionately peripheralward.

The measurements of the so-called "rod-free area" are:

Fovea Centralis ...	0·24 to 0·3 mm.	in diameter	(55′ to 70′)
Rod Free area ...	0·80 mm.	,, ,,	(3°3′)
Macula	1·0 to 3·0 mm.	,, ,,	(4° to 12°)

In order to fall within these areas, an object at a distance of 6 metres would have to be:—

Fovea Centralis ...	9·6 to 12 mm.	in diameter.
Rod free area ...	32 mm.	,, ,,
Macula	40 to 120 mm.	,, ,,

In order to stimulate an individual cone 0·004 mm. diameter, an object must be 1·7 mms. at 6 metres, which is the exact size of each element of a Snellen's 6/6 test-type letter.

An examination was made at 6 metres with the 1 millimetre aperture of the lantern of 313 colour defectives who had been classified on the 5 mm. aperture.

139 had been passed as "safe." 29 (20·9 per cent.) of these failed to recognise the red or green, or both the red and green 1 millimetre aperture.

174 had been considered as "unsafe." 77 (36·8 per cent.) of these were able to recognise both the 1 millimetre red and green.

TABLE VIII

Comparison of the effect of Aperture Sizes.

LANTERN			COLOUR
Classification of *Cases using the* 5 mm. aperture	Recognised 1 mm. aperture	Failed on 1 mm. aperture	Analysis of Failures
C.D. Safe ... 139	*97 (79·1%)*	*29 (20·9%)*	Red ... 1 *Green ... 26* Red and Green 2
C.D. Unsafe 174	77 (36·8%)	110 (63·2%)	Red ... 11 Green ... 79 *Red and Green 20*
Total ... 313	174 (55·6%)	139 (44·4%)	139

This supported the contention that although 1 millimetre colour targets are of undoubted value in campimetry, such small apertures *are of questionable value in examinations of colour-vision outside* the laboratory.

Granit supports this view when he states, "a reduction of area of the visual object, which is known to lead to disappearance of its *colour with maintained brightness distribution, must do so because* the "small" stimulus merely has a chance of hitting upon the *common dominators.*

Experimental evidence, however, shows that the recognition of a small colour light is subject to considerable individual variation.

In a series of papers published in the *Journal of Physiology* (1946), Hartridge has reported his experiments in micro-stimulation in support of his postulate that the foveal cones are frequently found grouped together into clusters by "random distribution" like the coloured starch grains on the Lumiére screen.

It is not easy to see how microscopic fixation can be retained for these experiments when the eye has a constant involuntary movement over two minutes of arc of 50 per second.

These particular investigations terminated because new lanterns which were obtained had apertures varying from 1 millimetre to 0·5 millimetre and it was not possible to standardise the results.

This fact was not discovered until one group of 66 subjects

gave figures which were below the average. Only six of them were able to recognise standard green and it was found that the aperture was only 0·75 millimetre in diameter, the small difference in size being apparently of much importance.

Successive contrast

In his discussion of after-images, Edridge-Green states, "the results obtained by successive contrast are similar to those obtained by simultaneous contrast, only they are more satisfactory and decided."

When a colour is looked at for a time, the sensitivity toward that colour is lowered and the eye becomes more sensitive to the complementary. In fact, when a coloured light is concentrated upon for some minutes the complementary becomes so insistent that the original light is seen with difficulty.

If a colour-vision examination is performed slowly, successive contrast is likely to complicate the findings. It was noticed, however, that this phenomenon was less in those with normal colour vision.

Successive contrast was investigated in 415 subjects whose colour vision was abnormal. 217 (61·0 per cent.) of these were "unsafe" and 139 (39·0 per cent.) were "safe."

Using the 5 mm. aperture, standard yellow was shown rapidly after standard red, then standard green. 356 (85·8 per cent.) demonstrated the phenomenon of successive contrast.

Of the total cases, 172 were unable to recognise standard yellow after standard red, whereas only 29 failed to recognise it after green. The percentage of 50 per cent. "safe" and "unsafe" was the same in each group.

155 subjects were unable to recognise yellow after both red and green. 78 per cent. of these were "unsafe."

TABLE IX

Lantern Classification	Successive Contrast after 5 mm. aperture showing			No Successive Contrast	Total
	Red	Green	Red and Green		
Safe ...	92 (53·5%)	13 (44·8%)	34 (22·0%)	13 (22·0%)	152
Unsafe ...	80 (46·5%)	16 (45·2%)	121 (78·0%)	46 (78·0%)	263
Total ...	172	29	155	59	415

It is interesting to note among those who were " safe " according to the single lantern, the proportion (22·0 per cent.) who were unable to recognise yellow after both red and green was the same (22·0 per cent.) as those who showed no successive contrast.

During this investigation it was found that whereas the phenomenon of successive contrast is exaggerated in those with defective colour vision (Guttmann, Edridge-Green) it was not marked in the normal. A number of men were found who apparently conformed to Edridge-Green's heptachromat. They read with ease the figures in the books of test-plates " not usually seen by normal persons," and after regarding the 5 mm. aperture for some time they made no mistake whatever in naming a 1 mm. yellow or white when it followed; their successive contrast was minimal.

It was not the purpose of these tests to examine such cases further.

Simultaneous contrast

Guttmann's seventh postulate, that anomalous trichromats have a marked difference in simultaneous contrast effects as compared with the normal, is supported by Edridge-Green, who states that simultaneous contrast is not pronounced in the normal person, but the phenomenon is exaggerated to the dichromat.

TABLE X

Contrasting colours	Apparent Move
Red and Blue Green	Becomes more intense Inclines to blue
Orange and Green-blue	Inclines to red Inclines to blue
Red and Orange	Inclines to rose red Inclines to yellow
Red and Yellow	Inclines to rose red Inclines to green
Red and Yellow-green	Inclines to rose red Inclines to pure green
Yellow and Green	Inclines to orange Inclines to blue

When two spectral colours are presented simultaneously, each colour appears to be moved toward that end of the spectrum further away from the other colour. Green and rose, also red and violet appear to be exceptions.

He goes on to say, " two colours, which have not changed in the slightest degree to the normal-sighted, on being contrasted have apparently altered very considerably to the colour-blind. As an example of this, let us take a deep yellow, a bright red and bright green. To the normal sighted the yellow will be altered very little by comparison with the red or the green, but a three unit would say that the colour was green when contrasted with red, red when contrasted with green."

The effect of simultaneous contrast was investigated by showing to the candidate two colour perception units 10 inches apart at a distance of 20 ft. The following pairs of colours were shown, using the 5 mm. apertures, in any combination.

Two standard green
Two standard red
Two standard yellow

A group of 52 subjects whose colour vision was suspected who had been investigated thoroughly by other means, were examined. Of 30 who had passed successfully a single-light test, 21 made errors when shown two simultaneously.

TABLE X1

Classification according to *Single* Colour Perception Unit (5 mm.)			Classification using *Two* Colour Perception Units (5 mm.)	
Unsafe	22	Red-blind ... 15 Green-blind ... 6 Red and Green-blind 1	Unsafe	22
Safe	30		Unsafe ... ... Safe	21 9
Total	52		Total	52

In this group of suspects, approximately 42·3 per cent. were considered unsafe with the single light, 82·7 per cent. with the double light.

This finding indicated that the single-light lantern was not sufficiently accurate in the separation of the " dangerous " from the " non dangerous," for a candidate could not be considered safe who was unable to recognise two 5 mm. signal lights at a distance of 20 feet.

A considerable number of lanterns was made until eventually it was found that three coloured apertures, shown simultaneously, constituted the most sensitive test and a lantern was constructed which would show any combination of reds, greens, and yellows, using apertures 10 millimetres in diameter, with variable illumination.

The method of examination was as follows: the lights in the consulting room were lowered but the room was not made dark enough, or the test continued long enough, for the patient to become dark-adapted. By means of the lantern rheostat the light was increased fairly rapidly until the three apertures were distinctly seen. At this illumination the patient was asked to name the colours of the lights as they were shown to him in groups of three.

It is considered that, under these conditions, any single error in the naming of the colours shows that the man's colour vision is unsafe.

137 subjects who had made errors in reading the Ishihara plates were classified on the single-light lantern. 28 made no mistakes and were assessed " safe."

These 28 were then tested on the new lantern and 19 made errors, an increase of 13·87 per cent. in those assessed " unsafe."

TABLE XII

	Single-light Lantern	Multi-light Lantern
Safe ...	28 (20·44%)	9 (6·57%)
Unsafe	109 (79·56%)	128 (93·43%)
Total ...	137	137

The most common error was to miscall standard yellow when it was shown together with either reds or greens, or even yellows.

The next common error was to call one of a trio of reds or greens, " yellow."

If the results of the single, double, and treble light examinations are compared, it is found that the sensitivity of the test increases.

TABLE XIII

Classification of colour defectives examined by three types of lantern

Lantern	Colour Defective Safe	Colour Defective Unsafe
Single Aperture	31·5%	68·5%
Double Aperture	17·3%	82·7%
Treble Aperture	6·57%	93·43%

It should be observed that the percentage of defectives by the double aperture is almost identical to the Ishihara.

This series of tests is evidence against the use of the single-aperture lantern and indicates the need for a fuller investigation of the anomalous trichromats hitherto considered as " safe."

Conclusions

1. This paper reports the investigation into the colour vision of 40,380 men, of whom 1,152 (3 per cent.) were safe, and 1,083 (2·5 per cent.) were unsafe.

2. Tests by colour plates, although more sensitive than tests by a single-lantern, are unreliable and differ with different series of plates.

3. The use of small apertures serves no useful purpose in the examination of colour-vision in the consulting room (Table VII).

4. The standard single-light lantern is not sensitive enough for discriminating between the " normal " and the " unsafe " (Table XII).

5 A triple-light lantern with controlled illumination is the most sensitive and reliable apparatus for demonstrating defects of the colour sense in the consulting room (Table XIII).

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A SURVEY OF ESOPHORIA AND CILIARY SPASM*

BY

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IT is with diffidence that I venture to write about ciliary spasm. Irregularities of the accommodative state seem to be on the increase and in some way connected with the post-war period, and patients are being referred in quite considerable numbers for orthoptic treatment.

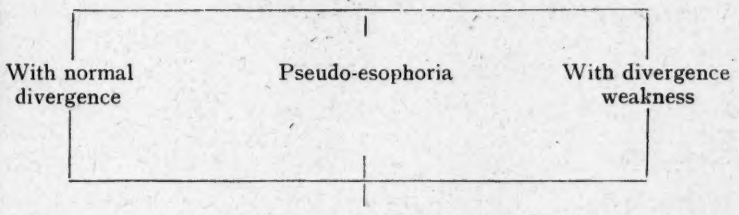
I thought, therefore, that it might be interesting to illustrate several cases which have responded well to treatment.

Before discussing ciliary spasm in detail, there are one or two straightforward cases of esophoria and old accommodative squint that have received great relief as a result of treatment, and show rather clearly the different categories into which these particular forms of ocular muscle imbalance seem to be divided.

In the four groups shown in Table 1 we get quite a wide variation of the initial defect *i.e.*, esophoria.

TABLE I

ESOPHORIA



1. *Esophoria with normal divergence.*—As is well known, this type of case is found to respond quickly to treatment and the amount of imbalance, though sometimes quite large, entirely disappears.

2. *Esophoria with divergence weakness.*—On the contrary, those in this group obtain relief from symptoms and are able to carry on their occupations successfully but owing to a divergence weakness a considerable amount of imbalance remains. Operation follows in these cases if the symptoms recur.

3. *Pseudo-esophoria.*—This is a state which is associated with

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ciliary spasm and will therefore be referred to in the second part of this paper. It is interesting to note, however, that the imbalance is very difficult to determine owing to the overaction of the ciliary muscle and the readings vary from day to day according to whether the accommodation is relaxed or working to excess. The variation can be as much as 10△ of esophoria or exophoria, and an examination on one or two consecutive days may be necessary before a decision can be made.

4. *Esophoria associated with old accommodative squint.*—In adults very severe symptoms can be experienced over a period of many years, and these can only be relieved by orthoptic treatment. The age factor is no great barrier providing binocular single vision is present, and the general health good. Dramatic changes in the

ESOPHORIA WITH NORMAL DIVERGENCE

Case 1. Age 62 years

Correction. R. + 1·25 D.sph., L. + 0·75 D.sph., Distance
R. + 3·75 D.sph., L. + 3·25 D.sph., Reading

	Before Treatment	*After Treatment*
Visual Acuity	R. 6/18, L. 6/18	R. 6/6, L. 6/6
B.V.A.	6/18, pt.	6/5
Worth's Lights	5	4
Maddox Rod	9△ esophoria	Orthophoric
Maddox Wing	3° esophoria	Orthophoric
Livingston Binocular Gauge	Convergence 12 cms. Accommodation 15 cms.	6 cms. 12 cms.
Synoptophore Angle ...	+ 8°	0/ + 4°
Fusion	+ 4°	0°
Adduction	8°	35°
Abduction	3°	5°
Stereoscopic Vision ...	Poor	Good

Number of Treatments 6. All tests taken with distance glasses

well-being of these patients can be noted and they state that their lives have been completely changed as a result of restored muscle balance.

In illustration of these points the following cases which fall into the several categories previously outlined have been chosen. All have been re-examined after treatment at different periods up to 2½ years and only one has had to receive a further course.

CASE NO. 1. Aged 62 years.—This patient complained of having suffered from severe headaches and giddy attacks for the past ten years, and was found to have a considerable amount of esophoria which caused her to get diplopia occasionally when tired. Being a case with normal divergence the imbalance was completely eliminated by treatment, with relief of all symptoms.

CASE NO. 2. Aged 15 years.—This child was said "to squint at meal times" and complained of headaches and diplopia when

ESOPHORIA WITH DIVERGENCE WEAKNESS

Case 2. Age 15 years

Correction. Nil

	Before Treatment	*After Treatment*
Visual Acuity	R. 6/4, L. 6/4	R. 6/4, L. 6/4
B.V.A.	6/4	6/4
Worth's Lights	5	4
Maddox Rod	12Δ esophoria	6Δ esophoria
Maddox Wing	I–11	01–3
Livingston Binocular Gauge	Convergence 14 cms. Accommodation normal	6 cms. normal
Synoptophore Angle ...	+ 6°	+ 1°
Fusion	+ 4°	0°
Adduction	Nil	20°
Abduction	4°	6°
Stereoscopic Vision ...	Too much suppression	Good

Number of Treatments 20

tired. The esophoria lessened somewhat and the symptoms disappeared, but there was evidence of divergence weakness. She has since commenced to study medicine and there is every likelihood of further treatment being needed, and possibly an operation.

CASE NO. 3. Aged 51 years.—A history of squint from early childhood was given here, and severe symptoms such as blurred vision, occasional diplopia, and acute headaches, had been experienced for the past 15 years. The prisms were removed after the first twelve treatments and at the conclusion of the second course complete relief from symptoms had been established. It will be seen, however, that the amount of esophoria remained at 12 degrees. An examination after a lapse of 2½ years found the patient quite comfortable and the measurements the same as at the last visit.

ESOPHORIA ASSOCIATED WITH OLD ACCOMMODATIVE SQUINT

Case 3. Age 51 years

Correction. R. $\frac{+ 1{\cdot}0 \text{ D. sph.}}{+ 0{\cdot}5 \text{ D. cyl.}}$ L. $\frac{+ 0{\cdot}5 \text{ D. sph.}}{+ 0{\cdot}25 \text{ D. cyl.}}$ 3 prism, B.O., E.E.
+ 2·0, added for reading

	Before Treatment	*After Treatment*
Visual Acuity	R. 6/5, L. 6/6	R. 6/5, L. 6/5
B.V.A.	6/5	6/5
Worth's Lights	5	4
Maddox Rod	20△ esophoria	12△ esophoria
Maddox Wing	3°–11 esophoria	Orthophoric
Livingston Binocular Gauge	Convergence 12 cms. Accommodation 23cms.	6 cms. 18 cms.
Synoptophore Angle ...	+ 15°	+ 4/8°
Fusion	+ 10°	0°
Adduction	15°	30°
Abduction	5°	10°
Stereoscopic Vision ...	Slow	Good

Number of Treatments 24. All tests taken with distance glasses

CASE NO. 4. Aged 42 years.—Here again a history of squint from four years of age with similar symptoms over a period of many years culminating finally in inability to read for more than five minutes at a time. A marked convergence insufficiency was shown at first, but later, after the removal of the prisms and a reduction of the correction, the true esophoria was revealed and treatment as for accommodative squint reduced the angle of convergence from 15° to 3° without glasses with relief of symptoms. When communicating some months later with this patient he said he had been away on holiday and had read a detective book per day.

ESOPHORIA ASSOCIATED WITH ACCOMMODATIVE SQUINT

Case 4. Age 42 years

Correction. Was wearing R. $\frac{+3{\cdot}75 \text{ D sph.}}{+1{\cdot}50 \text{ D cyl } \searrow 115^\circ}$ L. $\frac{+3{\cdot}50 \text{ D sph.}}{+1{\cdot}75 \text{ D cyl. } \swarrow 65^\circ}$

Was ordered R. $\frac{+2{\cdot}0 \text{ D sph.}}{+2{\cdot}0 \text{ D cyl. } \searrow 115^\circ}$ L. $\frac{+3{\cdot}0 \text{ D sph.}}{+1{\cdot}50 \text{ D cyl. } \swarrow 65^\circ}$ 2 prism BOEE

	Before Treatment	*After Treatment*
Visual Acuity	*R. 6/6, L. 6/4*	*R. 6/6, L. 6/5*
B.V.A.	*6/24*	*6/6*
Worth's Lights	*5*	*4*
Maddox Rod	*12△ esophoria*	*4△ esophoria*
Maddox Wing	*4°–6 exophoria*	*01°–3 esophoria*
Livingston Binocular Gauge	*Convergence 12 cms. Accommodation 18cms.*	*6 cms. 13 cms.*
Synoptophore Angle ...	*+ 15°*	*+ 3°*
Fusion	*+ 12°*	*+ 1°*
Adduction	*5°*	*40°*
Abduction	*5°*	*5°*
Stereoscopic Vision ...	*Good*	*Good*

Number of Treatments 24. All tests taken with distance glasses.

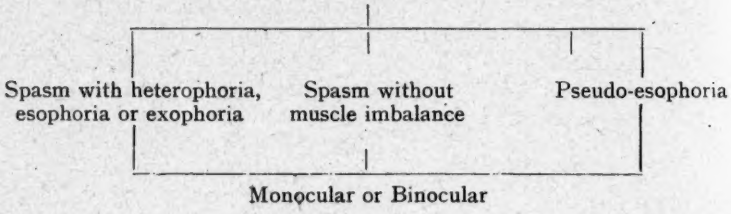
Ciliary spasm

This condition, which can be monocular or binocular, is referred to by Air Commodore Livingston in his paper in 1934 entitled "Heterophoria in Aviation" in which he tells us of "these complicated cases showing added spasm."

These under discussion have acquired a spasm for one reason or another, each one in a slightly different way and they fit into the various categories outlined in Table II.

TABLE II

CILIARY SPASM



It is surprising to find how many variations there are, and encouraging to realise what an immensely interesting and wide field they offer to orthoptists for treatment. That these patients in some instances suffer intensely there can be absolutely no doubt. On the other hand a young person in his teens can be suffering from a marked ciliary spasm of long standing and apart from noticing the blurring, of objects in the distance, can be otherwise completely ignorant of the fact.

The symptoms vary considerably, but amongst those complained of are :—

1. Blurred vision in the distance.
2. Rapid lowering of visual acuity.
3. Pain in the centre of the eyes.
4. Frontal headaches.
5. Sickness.
6. Tensing of ocular muscles generally.

At first it was thought best to rest the ciliary muscle by application of atropine before commencing orthoptic treatment, but latterly it has proved far more satisfactory to carry out both forms of treatment simultaneously. The reason for this is that if the spasm is of long standing and therefore a very strong habit an unconscious effort will be made by the patient to overcome the

atropine, and relaxing of the muscles generally by orthoptic treatment helps to counteract this. For the same reason it has been found more helpful to give ½ per cent. atropine b.d. rather than 1 per cent. as patients themselves in the latter case have complained of the effects of the atropine wearing off towards the end of the day with the consequent return of symptoms and renewed blurring of vision.

Unless any discomfort or irritation is experienced, with the approval of the ophthalmic surgeon, atropine is continued for as long as four to five weeks according to the severity of the case and orthoptic treatment is given twice or three times a week.

Where patients hold appointments, naturally this method of treatment necessitates sick leave, but the attempt to relax the abnormal fluctuations which occur in a ciliary spasm cannot be carried out economically by orthoptic treatment alone.

The following results will, I hope, help to illustrate the various forms of spasm treated and I will return later to the methods of treatment we have used to restore the normal accommodative state.

Two cases have been chosen to show spasm with esophoria, one being a myope and the other a hypermetrope with a very small correction. Both were young women who had had ocular symptoms for many years and finally both were compelled to give up good appointments on account of their general health being affected.

CASE NO. 5. Aged 26 years.—This patient had suffered for six years from severe headaches and a gradual onset of blurred vision accompanied by attacks of vomiting lasting for several days.

Her visual acuity deteriorated starting in July, 1939, as R. 6/9 L. 6/5. December, 1941, R. 6/9 L. 6/12. January, 1944, R. 6/36 L. 6/36.

Maddox Rod 8Δ esophoria and Maddox Wing 11—13 esophoria in glasses.

The binocular visual acuity at the commencement of orthoptic treatment in August, 1944, was 6/24 with glasses. This was restored to 6/9pt. after 24 treatments with complete relief of symptoms there being no further attacks of vomiting after the spasm had started to relax.

However, after a period of 2½ years the patient felt a slight tendency to return to the old habit of ocular tension and on examination it was found that the B.V.A. was 6/12. However, after a further course of treatment this was rectified. The cause of this relapse was thought to be due to a serious shock with the expected temporary setback to general health.

CILIARY SPASM WITH ESOPHORIA

Case 5. Age 24 years

Correction. R. $\frac{-5{\cdot}00 \text{ D sph.}}{-1{\cdot}00 \text{ D cyl.} \searrow 90°}$. L. $\frac{-4{\cdot}50 \text{ D sph.}}{-1{\cdot}00 \text{ D cyl.} \swarrow 90.°}$

	Before Treatment	*After Treatment*
Visual Acuity	R. 6/24, L. 6/24	R. 6/12, L. 6/12
B.V.A.	6/24	6/9 pt.
Worth's Lights	5	4
Maddox Rod	8△ esophoria	6△ esophoria
Maddox Wing	11°–13 esophoria	3°–5 esophoria
Livingston Binocular Gauge	Convergence 10 cms. Accommodation 16cms.	6 cms. 13 cms.
Synoptophore	+ 5°	0/+2°
Fusion	0°	0°
Adduction	30°	30°
Abduction	5°	7°
Stereoscopic Vision ...	Good	Good

Number of Treatments 36. All tests taken with glasses.

CASE NO. 6. Aged 26 years.—The history here revealed a tendency towards headaches for several years and the patient said she was often told at school that "her eyes were looking queer." Because she never felt very comfortable when reading, on volunteering for national service at 18 years she joined the Women's Land Army to avoid close work.

After several years' service she unfortunately met with an accident and fractured the base of her skull, and this precipitated acute ocular symptoms with occasional diplopia at the end of the day.

She was subsequently referred to an orthoptic clinic and found to have full ocular movements but 17° of esophoria.

She received 12 orthoptic treatments without relief and no reduction in the imbalance, although every effort was made to help her and her co-operation was excellent.

The surgeon decided that operation was not indicated at that stage but if no improvement occurred during the next 18 months he would reconsider the case.

In the meantime the patient was referred to me for investigation. I discussed the condition very fully with my colleague and we both agreed that there was some further problem yet to be discovered. This finally proved to be a ciliary spasm and by the time I saw the patient the vision had started to deteriorate to 6/18, either eye and binocularly.

Atropine was given together with treatment and the vision improved and the symptoms almost disappeared, the convergence angle being reduced from 20 degrees to 0 degrees as the result of the relaxing of the spasm.

Atropine was discontinued after a period of four weeks and very great care was taken to ensure that the patient completely understood how to relax her eyes and the importance of guarding against the possible return of the spasm.

CILIARY SPASM WITH ESOPHORIA

Case 6. Age 26 years

Correction Nil

	Before Treatment	*After Treatment*
Visual Acuity	R. 6/18, L. 6/18	R. 6/5, L. 6/5
B.V.A.	6/18	6/5
Worth's Lights	5	4
Maddox Rod	22△ esophoria	9△ esophoria
Maddox Wing	5°–11 esophoria	3°–9 esophoria
Livingston Binocular Gauge	Convergence 6 cms. Accommodation 6 cms.	6 cms. 12 cms.
Synoptophore	+10/20 after atropine 0	0/ + 4
Fusion	+10° after atropine 0°	0°
Adduction	20°	35°
Abduction	3°	5°
Stereoscopic Vision ...	Poor	Good

Number of Treatments 36.

She suffered acutely when learning to adduct and converge and frequently had attacks of vomiting after treatment.

The importance of helping the patient to persevere through these difficult phases of the treatment cannot be too strongly stressed and providing the health is normal in every other respect, orthoptic treatment should not be discontinued until they are overcome. In almost every case the same difficulties occur when trying to develop convergence without the return of the overaction of the ciliary muscle.

The effort to converge causes a very real disturbance which results either in nausea or in actual vomiting and one learns the necessity to watch the colour of one's patient and limit the time and method of treatment accordingly.

CILIARY SPASM WITH EXOPHORIA

Case 7. Age 17 years

Correction. R. $\frac{-8{\cdot}0 \text{ D. Sph.}}{-0{\cdot}50 \text{ D. Cyl.} \swarrow 60^\circ}$ L. $-8{\cdot}0$ D. Sph.

	Before Treatment	*After Treatment*
Visual Acuity with glasses	R. 6/18, L. 6/18	R. 6/9, L. 6/9
Without glasses	R. 6/36, L. 6/36	R. 6/18, L. 6/18
B.V.A. with glasses ...	6/9 pt.	6/6 pt.
Without glasses	6/36	6/18 pt.
Worth's Lights	5 crossed	4
Maddox Rod	$4^\triangle$ exophoria	$1^\triangle$ exophoria
Maddox Wing	$18^\triangle$ exophoria	$6^\circ - 8$ exophoria
Livingston	Convergence 14 cms.	6 cms.
Binocular Gauge	Accommodation 6 cms.	12 cms.
Synoptophore	-5° later -10°	$0/-2^\circ$
Fusion	-4°	$2^\circ -$
Adduction	5°	40°
Abduction	8°	10°
Stereoscopic Vision ...	Good	Good

Number of Treatments 30. All tests taken with glasses.

CASE NO. 7. Aged 17 years.—Here the spasm was slight, in fact only just commencing, and no symptoms at all were complained of in spite of a quite considerable amount of exophoria.

However, no attempt was made at first to reduce the exophoria but rather to relax the muscles, thereby revealing a further amount of imbalance, but at the same time restoring the visual acuity to normal. After reaching this stage the treatment proceeded as in the usual way for exophoria, observing closely for any return of the spasm on adduction and convergence. No atropine was used in this instance.

CASE NO. 8. Age 51 years.—We now come to a most interesting type of spasm, *i.e.*, that without any muscle imbalance of which only two cases have been referred for treatment, one having a binocular spasm and one a monocular. The first patient had been ill for some months and had suffered recently from alopecia areata.

CILIARY SPASM WITHOUT MUSCLE IMBALANCE

Case 8. Age 51 years

Correction. R. +1·75 D. Sph. L. +1·75 D. Sph.

	Before Treatment	*After Treatment*
Visual Acuity	R. 6/60, L. 6/60	R. 6/6, L. 6/6
B.V.A.	6/60	6/5
Worth's Lights	4	4
Maddox Rod	2△ esophoria	orthophoric
Maddox Wing	orthophoric	orthophoric
Livingston Binocular Gauge	Convergence 16 cms. Accommodation 10 cms.	6 cms. 19 cms.
Synoptophore Angle ...	0°	0°
Fusion	0°	0°
Adduction	20°	30°
Abduction	4°	6°
Stereoscopic Vision ...	Poor	Good

Number of Treatments 15. All tests taken without glasses.

She had had a long period of convalescence and came complaining of " going blind " saying that her vision had failed during the last four days. She also said that she had been compelled to hold her book nearer and nearer to her eyes.

As will be seen she could only see 6/60 E.E. and binocularly and, as was to be expected, was in a highly emotional state. She had rather poor stereoscopic vision, considerable suppression on the synoptophore, but otherwise showed no imbalance. The real trouble was detected when testing the accommodation on the Livingston Binocular Gauge which with and without glasses was 10 cms. instead of 19-20 cms. at the age of 51.

Atropine was prescribed and on account of the age of the patient, this was carried out under the personal supervision of the ophthalmic surgeon. It was discontinued after 12 days as the visual acuity had recovered to 6/9 and orthoptic treatment was given to strengthen the stereoscopic vision and relax the remaining spasm and thus restore visual acuity to 6/5 and the accommodation to 19 cms.

The patient made a rapid recovery to good health and has not had any return of the trouble.

CASE NO. 9. Age 14½ years.—The second case in this group which shows a monocular spasm, was quite different in that there were no symptoms complained of, merely the fact that the vision was " poor " in one eye.

Here we find the spasm tending to lessen over a period of time, rather than increase, as in most instances.

From the orthoptic point of view there was little to account for the condition except that the stereoscopic vision was very poorly developed indeed and there was marked suppression of the right eye with poor convergence.

The variation in the refraction, however, is of great interest as in 1944, the R.E. vision was corrected from 6/18 to 6/5 with R.E. −2·0 D. Sph. L. plane; 1945, the correction was reduced to −1.0 D. Sph.; 1946, the correction appeared to be the same, the vision being improved from 6/36 to 6/6 in glasses.

In all these previous instances, atropine had been given for three days prior to refraction.

In August, 1946, it was decided to continue atropine for fourteen days and the correction then accepted was R. + 0·5 D. Sph., L. + 0.5 D. Sph.

Atropine was continued for a further fourteen days, glasses were discarded and the right eye vision improved from 6/36 to 6/6 after twelve treatments.

This patient has now returned to school and will be seen at half

term to check the vision as we do not know as yet how well she will be able to stand up to close work.

It would seem that some patients suffering from ciliary spasm accept a myopic correction and to a certain extent are comfortable and, for a time, able to see better.

Sooner or later, however, the spasm increases, the symptoms become more acute and finally the glasses are no help.

CILIARY SPASM WITHOUT MUSCLE IMBALANCE

Case 9, Age 14½ years

Correction. April 1944, R. −2·0 D. Sph. Left Plane.
August 1946, R. +0·50 D. Sph., L. +0·50. D. Sph. After atropine for 14 days.

	Before Treatment	*After Treatment*
Visual Acuity with glasses	R. 6/5, L. 6/5	—
Without glasses	R. 6/36. L. 6/5	R. 6/6, L. 6/5
B.V.A. without glasses ...	—	6/5
Worth's Lights	4	4
Maddox Rod	orthophoric	orthophoric
Maddox Wing	orthophoric	orthophoric
Livingston Binocular Gauge	Convergence 6 cms. Accommodation 8 cms.	6 cms. 13 cms.
Synoptophore	0°	0°
Fusion	0°	0°
Adduction...	4°	20°
Abduction	2°	6°
Stereoscopic Vision ...	Very poor	Improved

Number of Treatments 12. To continue. All tests taken without glasses.

CASE NO. 10. Age 15½ years.—This condition often calls for atropine, as usually there is exophoria present although esophoric readings on the Maddox Rod and Wing may be recorded.

In this particular case it will be seen that the reading on the

Maddox Rod was orthophoric, the Wing 5-9 esophoric and Worth's Lights 5 homonymous.

After atropine the Maddox Rod showed 7△ exophoria, the Wing orthophoria, and the synoptophore varied from + 5 degrees to - 12.

Another case not illustrated here showed 10△ of exophoria and - 5° on the synoptophore on one day and on the following day 8△ of esophoria and + 10/15 on the synoptophore. These abnormal fluctuations lasted until atropine was given and the exophoria revealed was 10△.

PSEUDO-ESOPHORIA

Case 10. Age 15½ years

Correction. R. +4·25 D. Sph. −1·25 D. Cyl. L. +5·5 D. Sph. −1·75 D. Cyl.

	Before Treatment	*After Treatment*
Visual Acuity with glasses	R. 6/6 pt. L. 6/6 pt.	R. 6/5, L. 6/5
Without glasses	R. 6/12 L. 6/12	R. 6/9, L. 6/9
B.V.A. with glasses ...	6/5	6/4
Without glasses	6/9 pt.	6/6
Worth's Lights	5 homonymous	4
Maddox Rod	Orthophoric. After atropine 7△ exophoria	7△ exophoria
Maddox Wing	5° – esophoria. After atropine orthophoric	orthophoric
Livingston Binocular Gauge	Convergence 6 cms. Accommodation 10 cms.	6 cms. 12 cms.
Synoptophore	+2° −10 after atropine	0°
Fusion	0°	0°
Adduction	10°	28°
Abduction	4°	10°
Stereoscopic Vision ...	Good	Good

Number of Treatments 18. Still continuing. All tests taken with glasses.

Diagnosis

The history reveals repeated efforts to clear the print when reading finally resulting in the necessity to hold the book nearer and nearer to the eyes.

Sickness and giddiness accompany these extreme efforts causing an acute emotional state on account of the loss of vision.

General muscle tension, not only of the ocular muscles, adds to the aggravation.

In spasm the ciliary muscle is contracted causing excess accommodation. This state gives rise to the following indications.

1. Variable visual acuity with and without glasses.
2. Blurred vision in the distance.
3. Distant vision sometimes cleared by concave lenses.
4. Near point focus.
5. All usual signs of myopia.
6. Evidence on the synoptophore, *i.e.*, blurring, suppression, variable angle.
7. Readings on all tests vary from day to day.
8. The reading on the Livingston Binocular Gauge when testing accommodation are usually far in excess of the normal.
9. The resolving of all these states after the application of atropine.

Treatment

There are very few new ideas, if any, in the treatment of these cases, but, as previously mentioned, there is a considerable emotional element present and marked muscle tension generally, and the whole basis of the treatment is relaxation.

It might be argued that atropine does this for the patient but in many cases the brain is so accustomed to the bad habit that it makes every effort to overcome the effect of the atropine, and it is here that the orthoptist can be of greatest assistance in helping the patient to know what his eyes are doing, and by learning to relax, gradually place them in a position of rest.

In the final stages of treatment a very high standard is aimed at in the effort to restore the accommodative mechanism to normal and in doing so the smallest slides are used, and finally when teaching adduction the smallest Maddox fusion slide has to be mastered to at least 25-30 degrees.

Comparison between these figures and those required for a cure in convergence insufficiency would be unfavourable, and it must be remembered that the tendency for the ciliary muscle to overact again requires long and patient handling before a cure is obtained.

Summary

In conclusion, I think it is wise to assume that we are, as yet, only on the threshold of this extremely interesting application of orthoptic treatment and much will be revealed as time goes on.

The most important factor to stress is the necessity of testing the accommodation both binocularly and monocularly in patients of all ages and types excluding those actually suffering from strabismus.

CONTACT LENSES IN EXCELSIS*

BY

ALLAN H. BRIGGS

LINCOLN

IN 1938 I was consulted by a young man aged 22 years, who was anxious to join the Civil Air Guard. He had been rejected on several occasions at various centres owing to the fact that he was rather highly myopic. He was extremely interested in flying, and very keen to learn, but his myopia had proved an insuperable obstacle. He was anxious to know of any form of treatment, operative or otherwise, which might overcome his difficulty; and the only possible course I had to suggest was contact lenses. He was very willing to bear the expense and to try the experiment and I accordingly placed him in touch with the contact lens centre. His visual acuity was: R.V. c—8·0 D.sph. and—1·0 D.cyl. axis 170° = 6/6. L.V. c—4·0 D.sph. and—2·0 D.cyl. axis 170° = 6/5.

I heard later that he had been successfully fitted with contact lenses and had presented himself for medical examination again, without divulging that he was wearing contact lenses, and had been passed without their presence being suspected.

I lost touch with him during the war period, but was able to establish contact with him again recently after he had been demobilised. I was anxious to learn what had transpired, and I do not think I can do better than quote his letter to me: " . . . Briefly I enlisted in the R.A.F. in a ground trade in February, 1940, but transferred to flying duties during the following year. I had the usual medical examination, and was posted to Canada and America for training as a pilot. My lenses were not detected either at this medical Board, or at two subsequent Boards I had in America and upon my return to England. The American Board carried out

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by American doctors, was if anything, more thorough than those conducted by R.A.F. medical officers.

" After I returned from the States I was given some operational training and then posted to a Bomber Squadron as a pilot and completed quite a number of sorties over Germany. I was flying as Captain of Halifaxes at this time. Things went well until January, 1944, when my aircraft came into contact with some ironmongery over Berlin and as a result we had to abandon the thing by parachute some time later. I was extremely lucky and managed to get into France, there joining up with the Resistence people. After a few weeks with them things got a bit hot and I had to get out of France which I did by climbing the Pyr nees and getting into Spain. After a short period there I got back to England through the good offices of the Embassy in Madrid.

" Upon my return to England I had to undergo a further medical board—this was usual after one had become a casualty, but the results were the same as before. My medical category throughout was A.1b, and eyes 6/6. 6/6. The remainder of my service was as Captain of Transport aircraft (Liberators and Dakotas), first from this country to India, N. Africa, etc., and later I was posted to Australia for similar duties. I stayed there just over a year returning to England in April last, and from then until the end of my service on 1st of January this year (1947) I was employed as a test-pilot. My medical examination revealed the same result! I had no difficulty or trouble in the air irrespective of height or manœuvres, nor has service in tropical climates caused any discomfort. I managed to avoid dust storms, or the story might have been different—dust and grit is most unpleasant!

" I had two pairs of contact lenses, and always carried a spare pair with me; fortunately I never had cause to resort to the spare pair. None of my colleagues ever had the slightest clue that I wore these things, and the methods by which I contrived to wash and clean them at night, and replace them in the morning, are rather amusing. Nevertheless I always managed to carry out the operations without a great deal of difficulty. During the day I seldom had cause to remove them. The only occasions (fortunately rare) were when I had been a bit careless in putting them in, and had got a lash under them. I usually managed to find a quiet corner to remedy this trouble.

" I wear them for up to 17 or 18 hours a day, but normally for 16 hours. On two occasions only, when circumstances pressed a bit I wore them for periods of 30 and 36 hours respectively but I must admit my eyes were a bit 'tired' after this, and were bloodshot.

" I usually go to the contact lens centre about once a year for a check—the last time I went no change of power was necessary.

I am due to go any time now, and it may be that some slight change of power will be needed, as the 6/6 line was not quite so clear with one eye as it had been on previous examination, on my last examination by the R.A.F.

" I am not altogether severing all connections with flying, and I shall join an auxiliary Air Force squadron, or do some private flying. Consequently I still desire the secret to be kept and I know you will treat the whole matter with the confidence that it needs in the circumstances. Of course I have no objection to you using my experiences, provided my name does not appear connected therewith. Incidentally, even my wife does not know about them—I was married in August last year."

(The patient omits to mention in his letter that he was decorated for " courage, determination, and devotion to duty ").

It seems to me that there are certain important matters which arise out of this history.

(1) It seems clear that all the duties of an R.A.F. pilot can be carried out efficiently while wearing contact lenses. Even allowing for the fact that this may be an exceptionally successful case, and perhaps an exceptionally resolute patient, the position suggests that the visual acuity regulations for the services ought to be revised to show to what extent contact lenses will be permitted. If they give a satisfactory standard of visual acuity and can be worn for a reasonable length of time, it is difficult to see why they should not be allowed. If their use is permitted, it might permit many disappointed men who are otherwise physically fit, to have a second chance.

(2) It is manifest that contact lenses are not likely to be detected during a routine medical examination, even during one in which the estimation of visual acuity is regarded as of great importance. Ought any steps to be taken with regard to this? How far is it justifiable to aid and abet a patient in procuring contact lenses, knowing they are likely to be used to defraud a medical examiner, and how far is such a subterfuge unfair?

(3) The patient's letter demonstrates something of the future possibilities of contact lenses, both as regards their value as a visual aid and also as regards their difficulty of detection.

In conclusion, I should like to pay tribute to the courtesy and co-operation of the staff of the Contact Lens Centre, London, to whom all credit for the successful result in this case should be given.

ANNOTATION

Spare copies of the earlier volumes of the Transactions of the Ophthalmological Society of the United Kingdom.

We are informed by the Council of the Society that the printers have a large number of the earlier volumes of Transactions in stock. We understand that of the first 10 years (1880-89) there are more than 100 volumes. The younger members of the fraternity should embrace the opportunity of completing their sets. Only those who in the past have endeavoured to make good gaps in a long set of periodicals will know the difficulties and appreciate the excitement when a volume that at first sight seemed unobtainable comes in. The writer was once speaking on this subject to the late Mr. Treacher Collins and was told by him of the struggle he had to acquire the first volume of the Ophthalmic Hospital Reports, and of the high price he had to pay for it. Very early in our career we were lucky in purchasing vols. 2-7 of the Ophthalmic Hospital Reports at a reasonable price. We never saw a copy of the first vol. for sale but when our senior colleague retired from hospital he gave us his copies of vols. 1 and 2 which he had acquired for a shilling the pair at a sale of books at the beginning of the present century! In such a way do values and prices vary.

If the younger members of the Society do not rise to the bait the Council, in our opinion, would be well advised to consider either selling at a nominal figure or making a present of the earlier sets to such University Libraries among the allied countries of Europe as suffered damage and loss in the last world war. There must be many in Russia, France, Holland, Belgium, Norway and Denmark that would be glad to accept such an offer.

[The name and address of the printers is Headley Bros., The Invicta Press, Ashford, Kent. London office, 109, Kingsway, W.C.2. Telephone, Holborn 3309].

OPHTHALMOLOGICAL SOCIETY OF THE UNITED KINGDOM. ANNUAL CONGRESS

THE 67th Annual Congress of the Ophthalmological Society of the United Kingdom was held in the Department of Zoology, Glasgow University, on March 27, 28 and 29. The President of the Society, Professor A. J. Ballantyne was in the Chair. He delivered an address of welcome to the members of the Society and to visitors

from abroad, in particular, Dr. P. Merigot de Treigny (Paris), Dr. E. Godtfredsen (Denmark), Dr. J. Van Caneyt (Belgium), Professor W. H. Melanouski, Madame Schiss-Wertheimer and Madame Dallon. One hundred and eleven members attended the Congress.

The Presidential address, "De Senectute" was a clever and entertaining survey of the organic and functional changes that come to most of us whom the relentless march of time carries on to old age. Professor Ballantyne gave a general view of the tissue changes that occur with advancing years and in particular the familiar ocular disorders of cataract and presbyopia. The President gave useful advice about the explanation of these disorders to patients and the help this gave in their adjustment to such defects. He discussed the limitations of treatment and the possibility that new therapeutic agents might in the future postpone senile degenerative changes.

The subject of the Discussion was "Rhinology in Relation to Ophthalmology." The openers were Dr. J. Marshall, who dealt with the ophthalmological aspects of the subject, Mr. G. H. Howells described the part played by the Ear, Nose and Throat surgeon and Dr. R. McWhirter showed some admirable radiographs to demonstrate the importance of these in the diagnosis of inflammatory disorders, neoplasms and injuries of the orbit and adjacent structures. The openers had agreed not to discuss nasal sinusitis as a possible cause of retro-bulbar neuritis and intra-ocular inflammation.

The trend of the discussion was in favour of referring cases of orbital inflammation to the rhinologist. With his co-operation sulphonamide and penicillin therapy is carried on. It is preferable to drain into the nose an abscess originating from an infected sinus. If drainage is indicated through the orbit then it is best to do this in a plane between the orbital periosteum and the bone and not to open the orbital fascia.

The value of radiotherapy in the treatment of malignant neoplasms which have originated in the accessory nasal sinuses and have invaded the orbit was described and this treatment is preferred to surgical removal, particularly in the case of malignant neoplasms of the antrum.

For the exploration and removal of neoplasms deep in the orbit the relative merits of the trans-frontal approach and the lateral orbital access through Krönlein's operation were briefly discussed. There is no doubt that the former is indicated in cases when it is likely or possible that the neoplasm has extended beyond the confines of the orbital cavity and the latter is well justified when it is certain that the neoplasm is probably innocent, such as a cavernous haemangioma, neuro-fibromatosis and a cyst.

Opinion was more in favour of Toti's operation, dacryocystorhinostomy, or a modification of this for naso-lacrimal duct obstruction than of West's intra-nasal operation.

Orbital and ocular injuries as a result of nasal sinus operations were mentioned. Such calamities as penetration of the eye, orbital cellulitis, total ophthalmoplegia and division of the optic nerve are among these.

The subject rhinology in relation to ophthalmology afforded much material for discussion.

Other papers of interest were keenly discussed. These were:— "Induction of an Experimental Tumour of the Lens," Professor Ida Mann. "An Unusual form of Retinal Detachment (? cystic) seen in Children," Mr. F. A. Juler. "A Classification of Epiphora, with remarks on diagnosis and treatment," Dr. H. M. Traquair. "Some Aspects of Lid and Socket Repair," Mr. J. Scott Tough. "Bilateral Retinal Detachment Associated with Choroidal Cyst; a clinical and pathological report," Dr. J. Pendleton White and Dr. I. C. Michaelson. "Corneal Blood Staining," Professor A. Loewenstein. "Reiter's Disease," Mr. R. Lindsay Rea. "The Relation between Sjögren's Disease the Plummer-Vinson Syndrome, and Ariboflavinosis," Dr. E. Godtfredsen. "Observations on Holes at the Optic Disc," Mr. H. Neame. "Self-Inflicted Eye Injuries," Dr. L. B. Somerville-Large. "Vision During Glancing Movements of the Eyes," Dr. G. H. Bell and Dr. J. B. de V. Weir. "Vascular Disturbances in the Eye following Concussion Injuries," Dr. A. M. Wright Thomson. "Cysticercus Cellulosae of the Eye," Professor W. H. Melanowski. "Schnabel's Cavernous Atrophy," Mr. Eugene Wolff. "Some Aspects of Disease Affecting the Retinal Veins," Dr. A. J. Ballantyne and Dr. I. C. Michaelson.

A Clinical Meeting was held at the Glasgow Eye Infirmary. Twenty-seven interesting cases were shown and discussed.

The Annual Dinner of the Society was held in the Hall of the Royal Faculty of Physicians and Surgeons. Sir Andrew Davidson proposed the toast of the Society and the President replied. In a pleasing and witty speech Dr. S. Spence Meighan proposed the toast of the Guests. Dr. P. Merigot de Treigny and Professor Geoffrey B. Fleming replied to this.

Sir Stewart Duke-Elder's speech about the President was an admirable survey of Professor Ballantyne's qualities as a man and an ophthalmologist of international repute.

At the Annual General Meeting the Secretary read the Annual Report of the Society's activities. It is evident from the Treasurer's report that the Society's finances are in a sound and flourishing state.

A Trade exhibition of Ophthalmological Instruments and Appliances was held during the Congress.

BOOK NOTICES

The Surgery of Repair Injury and Burns. By D. N. MATTHEWS, MA., M.CH., F.R.C.S. Pp. 371, illustrations 198. Blackwell Scientific Publications, Ltd., Oxford. Price, 45/-.

The author has based the material of this book on his experiences as a surgeon in the R.A.F.V.R. where he has treated a large number of war injuries and burns, and has followed up these cases. He has omitted orthopaedic work, but inserted an account of the routine treatment of perforating wounds of the abdomen and thorax. The book deals mainly with the common war injuries which come within the province of the plastic surgeon. He describes the technique he favours. The chapter on the treatment of burns and shock is particularly well written. The book is well illustrated, many of the illustrations are in colour.

Chapter XIV deals very briefly with a few plastic operations for the eyelids and contracted socket. This chapter is not well done and contains a number of errors, some serious. The description of ptosis pays too little attention to the cause and the degree of ptosis and the manner in which these facts affect the treatment. The author does not comment on the undesirability of placing a load on the superior rectus, neither does he state in his description of passing fascia lata strips between the lid and the frontalis muscle for ptosis of what he calls neurogenic origin that any operation to raise the lid in III nerve palsy (ophthalmoplegia externa) condemns the patient to diplopia and so should not be done. His advice about placing a "pressure pad" over the eye after a ptosis operation is inviting a severe corneal abrasion. His histological knowledge is seriously at fault when he describes the tarsus as composed of "cartilage."

The best ptosis operations such as advancement and partial resection of the levator palpebrae superioris and Blascovics's operation are not mentioned.

It is difficult to understand why dacryocystorhinostomy is considered a plastic operation. It is evident from the author's description that he has never seen the operation done properly. The incision he advises is much too far forward, and with this it is little wonder that he is embarassed by haemorrhage from the angular artery and recommends that "it is wise to spend some time in identifying and ligating it before proceeding with the removal of the bone." There are many important technical details omitted, and the lacrimal retractor shown in Fig. 154 is so crude that it would damage the eye during operation.

We cannot agree that the raw area for paramedian tarsorrhaphy need be as long as 1 cm., nor that a Wolfe graft has poor vitality.

Stent mould pressed into the socket in socket reconstruction gives a deep irregular shaped socket with an uneven surface. It is better in the reviewer's opinion to use an acrylic mould made of the shape of the normal conjunctival sac after excision of the eye, in fact the shape of a prosthesis, and to attach to this the graft for insertion into the socket.

The book is well produced, the paper and illustrations are good. It is a useful and practical contribution to traumatic and plastic surgery.

Klinische und Erbbiologische Untersuchungen ueber die Heredoataxien. By T SJÖGREN. Acta Psych. et Neurol. Supp. 27. Copenhagen. 1943.

A survey of the literature concerning the hereditary ataxias shows that much difficulty is experienced in separating all the diverse clinical types. Numerous transitional forms, *e.g.*, between Strümpel's spastic degeneration and the Charcot-Marie neuromuscular atrophy, have been described. A recent and very thorough study of the subject has been made by Dr. T. Sjögren, whose work on the genetics of diseases of the central nervous system is already well known. The new monograph describes cases and their families discovered through the Swedish hospitals. All cases of Friedreich's ataxia, of Marie's ataxia and of other similar ataxias were collected. The material is fully analysed from the clinical point of view, details of family histories and geographical distribution of cases are given. There are numerous illustrations and tables. Altogether, 95 male and 93 female cases in 118 families are reported.

Dr. Sjögren divides his material into four groups and one subgroup. Groups I and IA contain cases of Friedreich's ataxia—those in 1A were less typical than those in I. The whole group contained 101 cases in 64 families. The frequency of this disease in the general population is estimated to be 0·0035 per cent. Fifteen per cent. of the cases were mentally defective. The mean age of onset in the typical Friedreich cases was 12 years and in the less typical cases, 15 years. A strong tendency was found for the age of onset to be constant in different affected members of the same family. This may indicate that, even within this selected group, there is more than one type of disease. All parents were unaffected. In six families the patients were first cousins and in three families they were more distantly related. This high consanguinity incidence coupled with the estimated familial incidence in sibships of about one quarter makes it highly probable that Friedreich's ataxia is recessively inherited. The possibility that there may be more than one allelic gene accounting for differences in onset age in different families is not discussed in the monograph nor is the material critically examined for evidence of partial sex linkage.

Group II contains 12 cases of transitional type in 12 families with mean age of onset of 50 years.

Sixty-four typical cases of Marie's ataxia in 35 families are classed in Group III. The mean age of onset here was 34 years, and in each family one parent was affected. There seems to be little doubt that the condition is dominant but, as with Friedreich's ataxia, affected members in the same family tend to have similar onset ages. The incidence of Marie's ataxia in the Swedish general population is estimated at 0·0014 per cent.

Dr. Sjögren's Group IV contains 11 cases of a mild hereditary type of ataxia, present from birth and lacking progressive tendency. Here the cause is almost certainly a dominant gene.

The method of combining genetical and clinical data before attempting to sort out different disease types is coming more and more into general use. The three types of data, clinical and pathological, statistical—age of onset and rate of progress of the disease—and family history material are all considered by Sjögren in the grouping of his cases. Though some transitional cases are still not fully accounted for, the method clearly enables great strides to be made towards the goal of defining the main types of nervous diseases and ascertaining the causal mechanisms behind them.

Les yeux et la vision des vertebres. ROCHON DUVIGNEAUD. Pp. 718, 500 illustrations. Paris: Masson et Cie. 1943.

It is a heartening thing to find so beautifully produced and so comprehensive a monograph appearing from Paris in 1943. Despite innumerable difficulties of paper shortage, permits, restrictions and set-backs of all kinds the doyen of French comparative ophthalmology has crowned his life's work with this outstanding publication and both he and Masson are to be congratulated. The death of the artist, M. Bessin, almost in the act of completing the superb illustrations, is a tragedy, since the completed work is as great a monument to his skill and industry as it is to its author's erudition and painstaking research.

The treatise deals with the gross and minute anatomy of the eyes of all the subdivisions of the vertebrates. It is written from the point of view of the human anatomist and ophthalmologist rather than the phylogenist and therefore the first 150 pages deal in detail with the structure of the human eye. Thereafter we are led from palaeontology, through vertebrate embryology to comparative anatomy of a most comprehensive type. The 500 beautiful illustrations form a collection only equalled by that of Gordon Walls and definitely surpassing the older monograph of Ovio. Throughout, the stress is on structure and accuracy of measurement and many of the data concerning the dimensions of the various vertebrate eyes are only available here. The work is essentially the result of painstaking

personal observation. Its insistence on structure, largely to the exclusion of speculations as to function, makes it a most valuable companion volume to that of Walls, with its phylogenetic and adaptational descriptions of function. Every ophthalmic library in the world will be incomplete without a copy.

Die Erkrankungen des Augenhintergrundes. By Dr. ADALBERT FUCHS. Franz Deuticke, Vienna. 1943.

In 1943 Adalbert Fuchs published a book in Vienna on Diseases of the Fundus, copies of which have now come to this country. The book is essentially a pathological treatise with an atlas of drawings of fundus diseases and drawings of the corresponding histological sections. It is divided in two parts. The first is a simple factual description of the commoner pathological conditions affecting the retina, the choroid or optic nerve; it is didactic in nature, without discussion and without references to the literature. The second part is a comprehensive atlas in which each case is fully reported and each picture described; many of the cases have already appeared in the literature. Some of the drawings are good, and some of them not convincing; for example, both the painting of the fundus and that of the microscopical section illustrating solitary tubercle of the nerve-head are lacking in sufficient detail to convey an adequate impression of the condition. Now that the technique of histological photography has reached so high a standard, it would seem that little is to be gained by semi-diagrammatic and somewhat artificial paintings which can convey little to the advanced student or the critical reader. At the same time the book, considering the date of its publication, is beautifully produced.

CORRESPONDENCE

ASTIGMATISM IN INDIANS

To the Editors of THE BRITISH JOURNAL OF OPHTHALMOLOGY.

DEAR SIRS,—Duke-Elder quotes Nordenson (1883) as proving the existence of physiological astigmatism, averaging 0·50 — 0·75 D., and being "with the rule" (the axis of least curvature not more than 30° from the horizontal) in 89 per cent. of a series of cases.

Refraction of Indian troops suggested that this rule did not apply to them, and so I have analysed 1449 consecutive cases of astigmatism refracted in my department. This includes all refractions; not only those for whom glasses were prescribed. The Indians were all adult males, the British included some military families.

BRITISH

Age	Total	All cases		Below 1·0 dioptre	
		With the rule	Against the rule	With	Against
Below 20 ...	49	28	21	8	8
20-29 ...	273	187	86	86	62
30-39 ...	135	83	52	39	36
40 and above	84	35	49	26	43
Totals ...	541	333	208	159	149

INDIAN

Below 20 ...	51	19	32	17	16
20-29 ...	367	151	216	91	156
30-39 ...	269	65	204	44	149
40 and above	221	49	172	36	119
Totals ...	908	284	624	188	440

While the British cases follow the "rule" much less closely than expected, the Indian actually show a majority against it. The total figures shown as percentages are:—

	Below 1·0 D.		All cases	
	With	Against	With	Against
British	51·5	48·5	61·6	38·4
Indian	30·0	70·0	31·3	68·7

With increasing age there is the usual increase of cases against the rule: the only keratoconus had 8 D. of astigmatism with the rule. These are two facts commonly quoted in support of the contention that physiological astigmatism is due to lid pressure on the globe.

The only factor which I can suggest as responsible for this inversion of the rule in Indians is trachoma, which is found in a majority of the soldiers seen in this part of India, and often in severe form. This is offered solely as a suggested explanation: I find no reference to the condition in the text-books available, and have no access to the literature.

I wish to thank the Commanding Officer, British Military Hospital, Delhi for leave to publish this note.

Yours faithfully,

R. A. D. CRAWFORD.
Major, R.A.M.C., Ophthalmic Specialist
AGRAM, BANGALORE,
INDIA COMMAND.

THE "NORMAL" IN THE SYNOPTOPHORE

To the Editors of THE BRITISH JOURNAL OF OPHTHALMOLCGY.

DEAR SIRS,—Referring to the paper by M. M. Lewis in your December issue and to N. Cridland's comments in the February issue, I would suggest that the latter's statement that ". . . . I do not think that there is, on the face of it, reason to suppose that the synoptophore and the Maddox rod measure different qualities of heterophoria" should not pass without comment.

I am not clear as to the meaning of the term "*qualities* of heterophoria" but, with regard to the degree of heterophoria that will be revealed, surely when the two eyes are dissociated so that the visual mechanism finds itself in an unusual state of balance, or imbalance, to deal with which it has no safe guide from its normal experience, it is to be expected that the position taken up by the eyes will fluctuate round about a mean position which may be definitely affected by the kind of apparatus that has been used to secure dissociation. This is to be expected especially when testing at near, when the whole mechanism is in a dynamic state and by suspending fusion we have eliminated the incentive for the governing innervation of the complex group of innervations that are in operation. I think that many practitioners will agree that this expectation is borne out in practice; for the results obtained,

especially in near testing, with the Maddox rod method do not in general agree with those obtained with the synoptophore.

Yours faithfully,

H. H. EMSLEY.

ALLIED INSTRUMENT MANUFACTURERS LTD.
20-24, PARKWAY, N.W.1.
March 13, 1947.

REMOVAL OF THE WRONG EYE

To the Editors of THE BRITISH JOURNAL OF OPHTHALMOLOGY.

DEAR SIRS,—I am grateful to Dr. Ascher for his letter on my paper on "Removal of the wrong eye." At the same time I do not follow his inference that the paper suggests that a case of this kind occurred in Elschnig's clinic either before or after 1908. Dr. Ascher's remarks about the meaning of "Soll" are, of course, correct, but seem to me to be irrelevant. I made no attempt at a literal translation as I thought that clumsy English would result and the free rendering I gave of the sentence in question conveys no implication of the kind Dr. Ascher indicates.

I have no information that such a case ever occurred in Elschnig's clinic but I am assured that Elschnig used to impress the dangers of this accident on his students, which suggests that he had heard of at least one case. The significance of his foot-note is clear: it means that between writing his text and correcting the proofs—otherwise why a foot-note?—he had learned of another case. I was careful to write "within his knowledge." The case may have occurred anywhere, possibly outside Austria altogether.

Cutting the eyelashes is only another method of marking the eye, though perhaps a better one than the use of adhesive plaster. The important point is that, for one reason or another, methods of marking the eye have proved unreliable.

I have for many years given up cutting the lashes in eye operations except sometimes a few at the lateral and medial ends of the upper lid. I doubt whether any advantage is gained by cutting even these.

Yours faithfully,

H. M. TRAQUAIR.

16, MANOR PLACE,
EDINBURGH, 3.
April 10, 1947.

FOUND—PATHOLOGICAL SPECIMENS

To the Editors of THE BRITISH JOURNAL OF OPHTHALMOLOGY.

DEAR SIRS,—A few days ago I was informed by the police that a package has been found in Lincoln, comprising a bottle containing two human eyes and a smaller bottle containing what appeared to be a crystalline lens, complete in its capsule.

These specimens appear to have been removed by an ophthalmic surgeon recently. How they came to be abandoned in Lincoln is at present a complete mystery and it seems possible that they may have been stolen from an ophthalmic surgeon's car and abandoned by the thief on discovering their nature. We have made local enquiries but have so far failed to discover any clue as to the owner of these specimens and I wonder if you would give publicity to the matter from your columns in the hope that the ophthalmic surgeon concerned may chance to hear of the matter in this way.

We should be very grateful if the rightful owner will get in touch with me, and on supplying particulars I should be pleased to arrange for the specimens to be handed over to him. It seems more than likely that these specimens were of some special value and we are anxious to restore them to their ower if possible.

Yours faithfully,

ALLAN H. BRIGGS.

3, LINDUM ROAD,
LINCOLN.
April 8, 1947.

THE USE OF UNIT CELLS IN THE LISTER MORTON OPHTHALMOSCOPE

To the Editors of THE BRITISH JOURNAL OF OPHTHALMOLOGY.

DEAR SIRS,—I have noticed that if I use unit 1½ dry batteries in my Lister Morton Ophthalmoscope, they often run down prematurely.

The following paragraphs explain the reason and give the remedy for this occurrence, which I am sure many users of the instrument must have also experienced.

The correct dry battery to use is, of course, the Ever Ready 1829, and this gives no trouble in use.

However, it is not so easily procurable as the standard 1½ volt unit cell, and the latter is, I think, most commonly used.

One of the features of the Lister Morton Ophthalmoscope made by Theodore Hamblin, Ltd., is a retaining ring of metal which is inserted at the upper end of the battery handle.

This ring retains the battery, when the head of the instrument is removed from the handle for insertion into the instrument case provided, and is a useful feature of the ophthalmoscope. Unfortunately the 1½ volt unit cells are so manufactured that the zinc casing, which is the negative pole, is exposed at the upper end of the cardboard insulating envelope, and can make contact with the retaining ring of the battery handle.

This causes a short circuit of the lower cell in the battery handle, and only happens when the handle is detached from the head of the instrument.

The remedy is either to depress the battery a little in its cardboard container and roll the edge of the cardboard over the upper rim of the zinc casing of the cell, or to glue a thin ring of cardboard as an insulator under the metal ring in the ophthalmoscope handle.

The correct battery for the ophthalmoscope, the Ever Ready 1829, is a two cell, 3 volt battery, enclosed in a single cardboard envelope the ends of which are rolled over, and so prevent the short circuit described when using two single cells.

Yours faithfully,

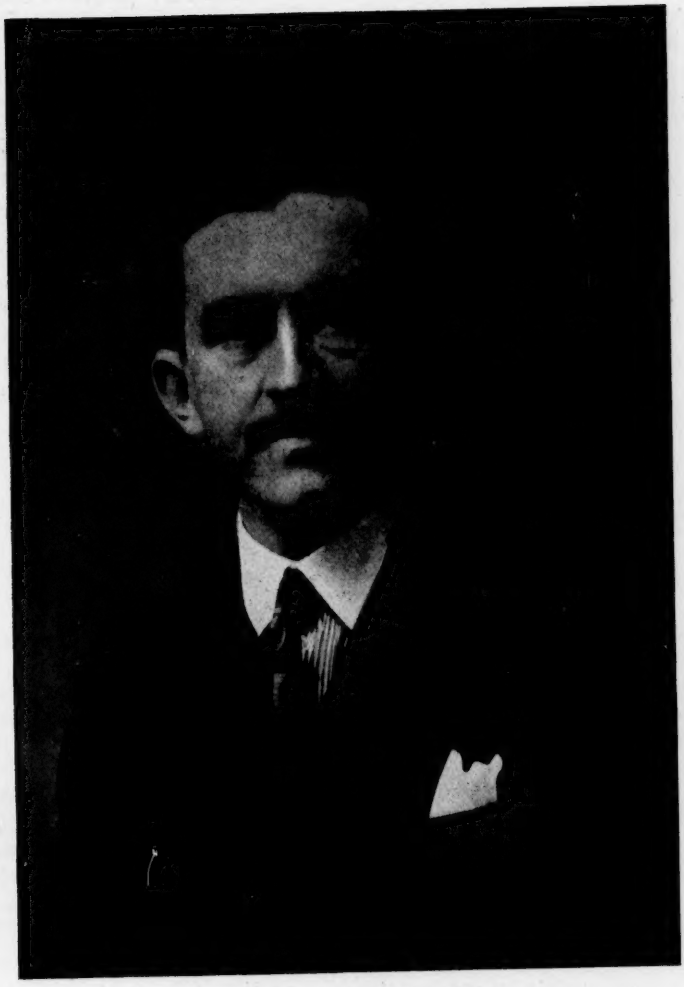
P. T. LEES.

7, ALBANY AVENUE,
BLACKPOOL.

OBITUARY

IAN STEWART McGREGOR

OPHTHALMOLOGY could ill afford the death of Dr. Ian Stewart McGregor on January 23 at the age of 43 years, when already he had shown great capacity as clinician, surgeon, teacher and original investigator. He graduated M.B. CH.B. at the University of Glasgow in 1927 and thereafter filled various resident appointments. His attainments were all the more remarkable in that his interests in ophthalmology began only nine years ago, previous to which he was in general practice on the Island of Bute. This experience in general medicine, however, so broadened his outlook and sharpened his judgment that he learned quickly and fastidiously from his colleagues and within a brief period was appointed Clinical Assistant at the Glasgow Eye Infirmary and Assistant Ophthalmic Surgeon to the Ophthalmic Institution of the Glasgow Royal Infirmary and obtained the Diploma of Ophthalmic Medicine and Surgery granted by the Royal Colleges in England. At the outbreak of war he was mobilised as a squadron leader in the R.A.F.V.R. where he served for two years. His release was requested in 1941 to fill a vacancy as Visiting Surgeon to the Ophthalmic Institution. He also acted



IAN STEWART McGREGOR

as Senior Assistant to the University Department of Ophthalmology. Despite the great demands of hospital practice in the war years he became a Fellow of the Royal Faculty of Physicians and Surgeons and of the Royal College of Surgeons in Edinburgh. He graduated M.D. in 1943, the subject of his thesis being the "effect upon the eyes of methyl alcohol poisoning." Self critical he published only after his precise staking of the known boundaries of ophthalmology. His publications included the following important papers:—

Orbital cellulitis from gas producing organisms. *Brit. Med. Jl.*, 1, 292-293, 1942.

Reticulin content and prognosis in malignant melanoma of uvea. *Arch. Ophthal.*, 30, 291-297, 1943 (with Hill, J.).

Study of histopathological changes in retina and late changes in visual field in acute methyl alcohol poisoning. *Brit. Jl. Ophthal.*, 27, 523-543, 1943.

Quinine blindness. *Lancet*, 2, 566-567, 1944 (with A. Loewenstein).

Bilateral partial ectasia of nerve head with peripapillary ectasia. *Brit. Jl. Ophthal.*, 28, 618-622, 1944.

Macular coloboma with bilateral grouped pigmentation of retina. *Brit. Jl. Ophthal.*, 29, 132-136, 1945.

Segmental movement of pupil. *Brit. Med. Jl.*, 1, 629-630, 1945.

Cyclic oculomotor palsy. *Jl. Neurol. Neurosurg. and Psychiat.*, 8, 22-23, 1945.

His scientific enthusiasm was tempered by the wider necessities. He was a dependable colleague, and his knowledge of the known limits and an imagination for the stretch beyond made his opinion on a case or problem sound and stimulating. This was often freshened by his piquancy of phrase. His patients were particularly fond of him, his keen mind and natural compassion rarely leading him astray in that proper blending of assessment and charity which they require. Deeply interested in literature and in the countryside he was a good example of what these, combined with an experience of general practice can do to perfect the interests and quality of a speciality. He was unmarried, and our very sincere sympathies go to his mother on her great loss.

NOTES

Honour

MR. COLVILLE MUIRHEAD has been elected an Hon. Member of the Societé belge d'ophtalmologie.

Appointment

Dr. P. H. BEATTIE has been appointed ophthalmic surgeon to the Norfolk and Norwich Hospital.

* * * *

National Society for the Prevention of Blindness.

THE National Society for the Prevention of Blindness announces the appointment of Dr. Franklin M. Foote to the position of Executive Director succeeding Mrs. Eleanor Brown Merrill who is retiring. Mrs. Merrill has been associated with the Society for more than twenty-five years and has served as the Executive Director for the past eight years. She formerly held the positions of Associate Director and Secretary.

Dr. Foote joined the Society's staff as Medical Director in 1946. He was formerly District Health Officer of the Kips Bay-Yorkville Health District of the New York City Health Department. Prior to that, he was Chief of the Division of Local Health Administration, Connecticut State Department of Health. Dr. Foote is Assistant Professor of Public Health and Preventive Medicine at Cornell University Medical College. Dr. Foote holds degrees of B.S., M.D., and Dr. P.H. from Yale University. During World War II, he served as a Major in the Medical Corps of the United States Army.

* * * *

White Oak Hospital, Swanley, Kent. 1944-46

DATA on admissions during 1944-45 are incomplete owing to the evacuation of the Hospital during part of each of these two years. For 1946 there were 222 admissions distributed as shown:

Blepharitis	79
Phlyctenular ophthalmia	109
Interstitial keratitis	4
Recurrent styes	16
Others	14
	222

In addition the Ophthalmia Neonatorum Unit which is still housed at White Oak, admitted 163 babies and 30 nursing mothers. Eight babies and eight mothers required re-admission.

* * * *

IIIrd Congress of Pan-American Ophthalmology

THE Congress will be held in Habana from February 22 to 28, 1948. Dr. Tomas R. Yanes is President of the Local Committee and will be assisted by members of the Sociedad Cubana de Oftalmologia.

* * * *

Corrigenda

IN Dr. Inglis Pollock's letter, p. 123, the following corrections should be made. Line 14, 303 should be 503. Line 15, Anglicum should be Anglicam and translation should be translatum. Line 16, pages should be folios. Line 17, 68 should be 136. Line 20, MMS. should be MS.